The Journal of Bone and Joint Surgery

The Official Publication of
The American Orthopaedic Association
The British Orthopaedic Association
The American Academy of Orthopaedic Surgeons
The Australian Orthopaedic Association
The Canadian Orthopaedic Association
American Society for Surgery of the Hand
New Zealand Orthopaedic Association
South African Orthopaedic Association

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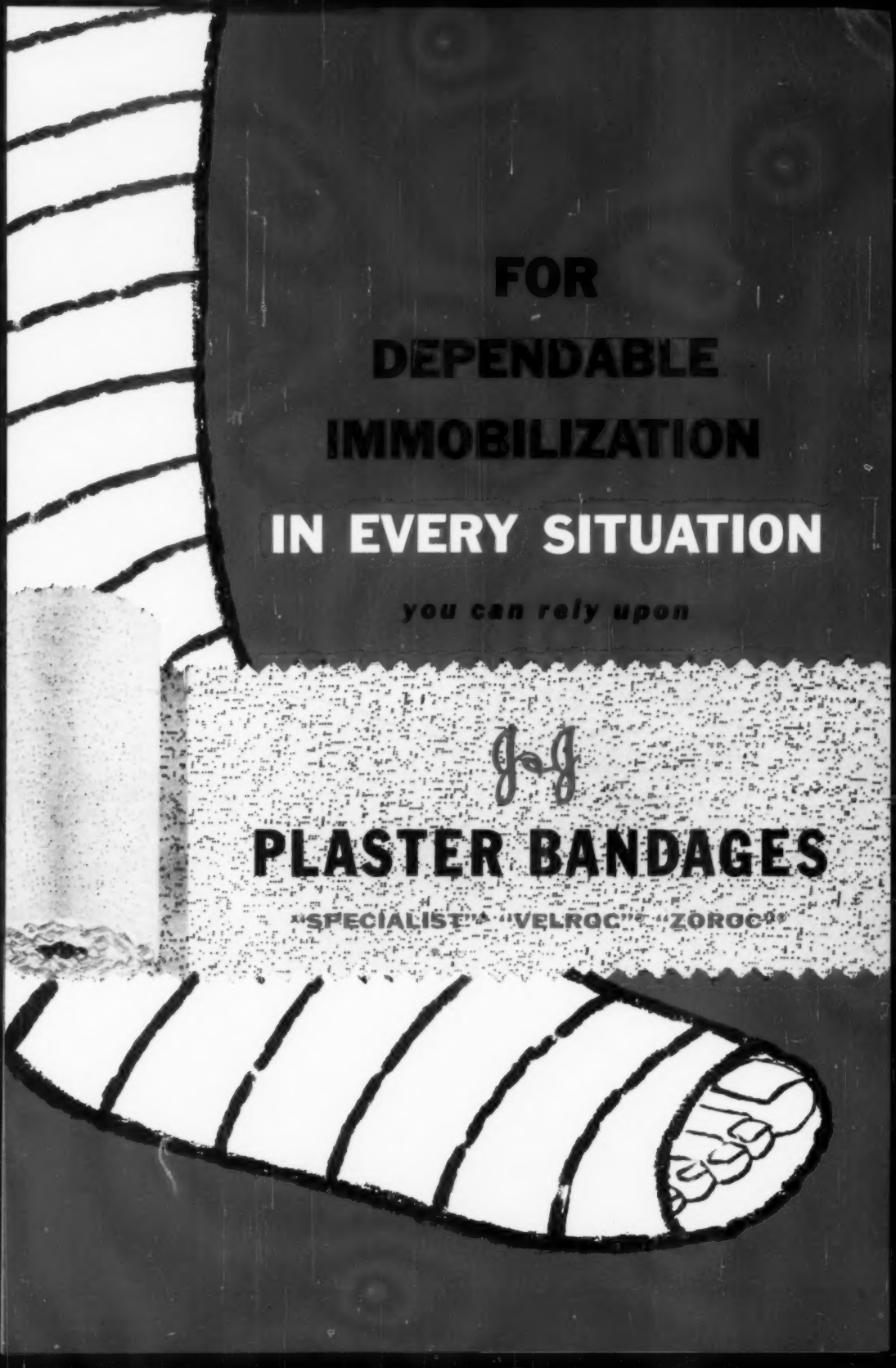
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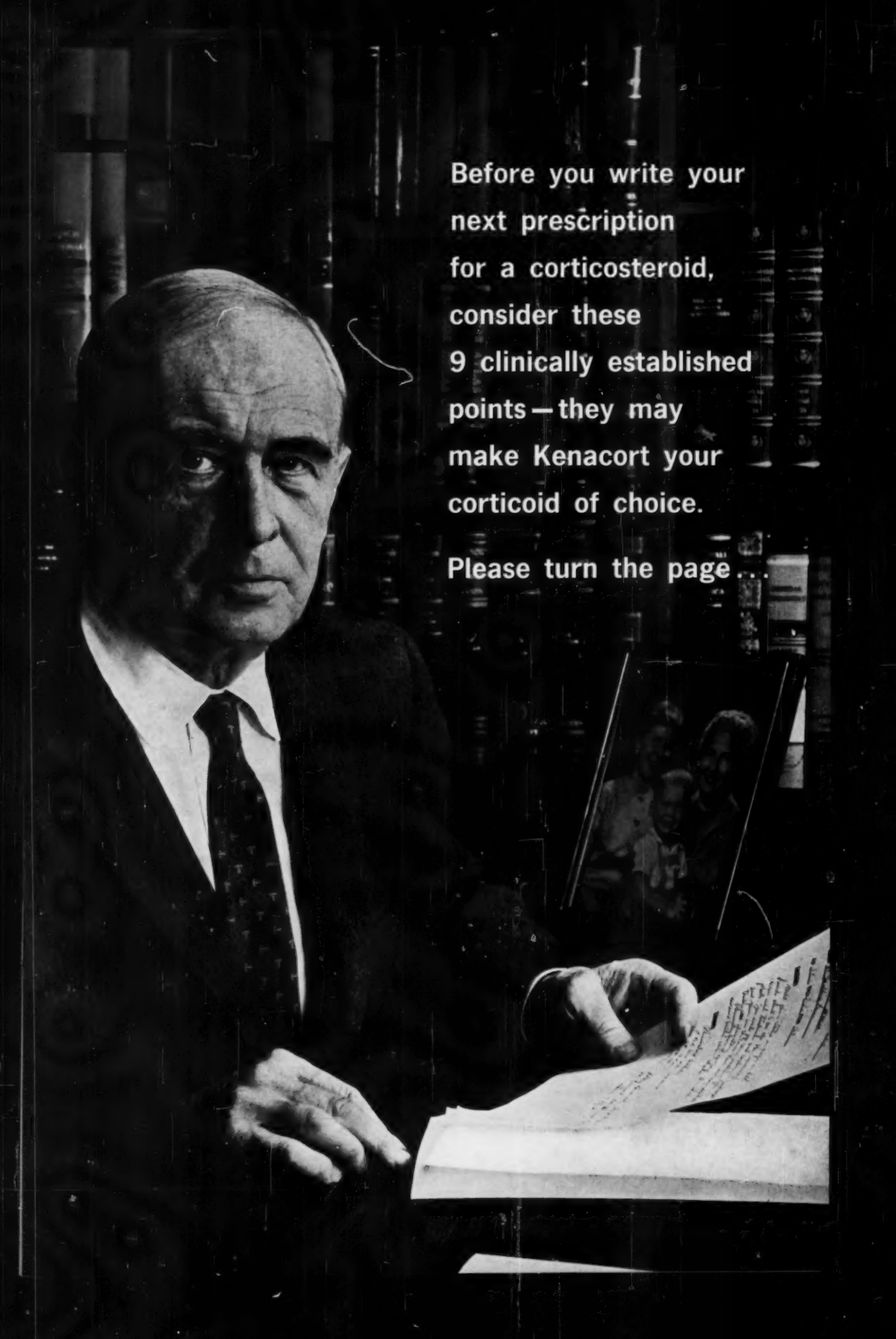
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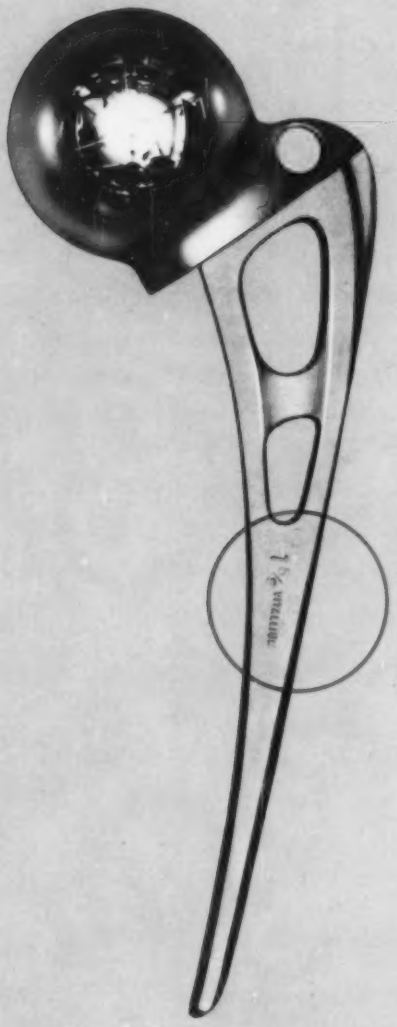

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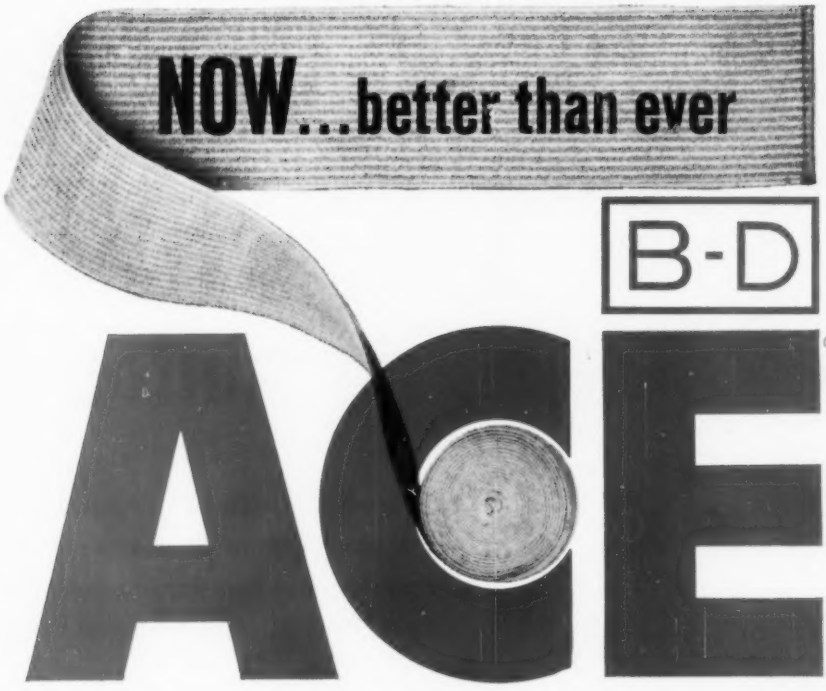

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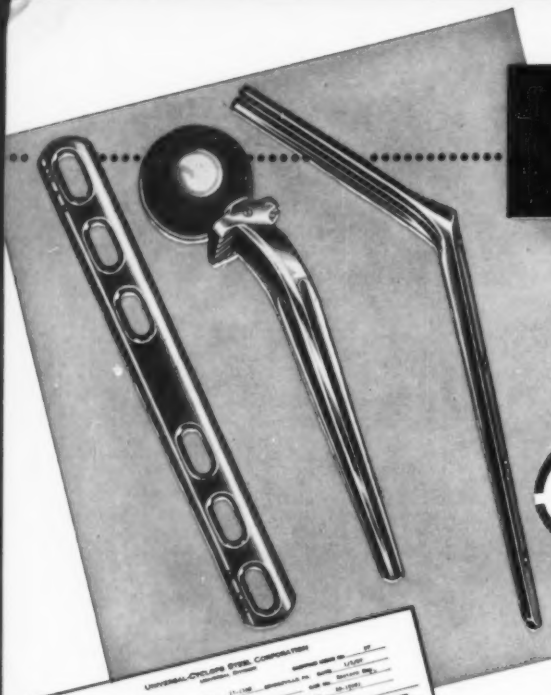

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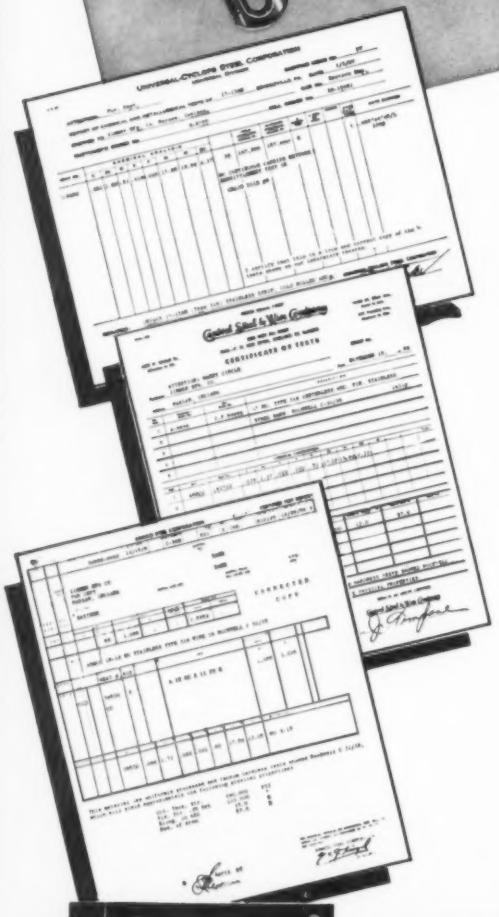

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SLOTTED PATTERN PLATES

No. 539

ADJUSTABLE INTERTROCHANTERIC PLATE

McLAUGHLIN TYPE

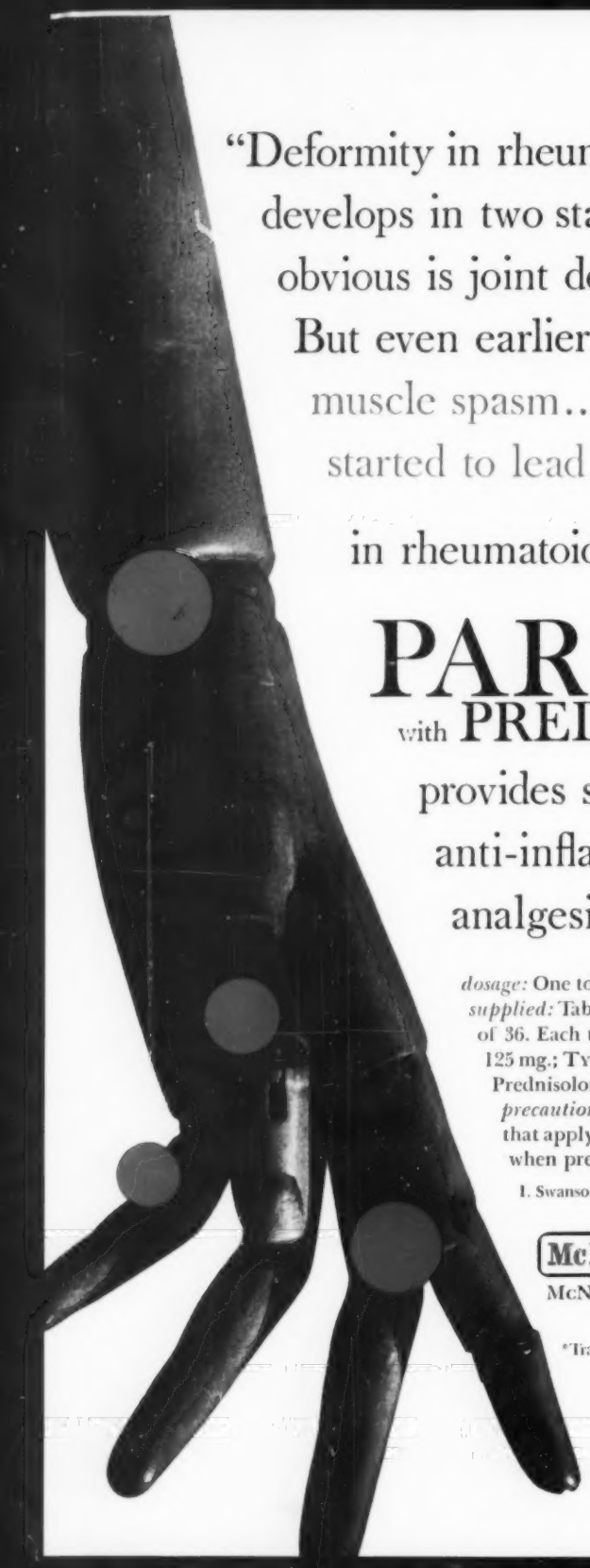
ELLIOT FEMORAL CONDYLE BLADE PLATE

No. 554

No. 560
(Nail Not Included)

ZIMMER MANUFACTURING CO. • WARSAW, INDIANA, U.S.A.

zimmer



"Deformity in rheumatoid arthritis develops in two stages. The most obvious is joint destruction. But even earlier... muscle spasm...has insidiously started to lead to deformity."[1]

in rheumatoid arthritis...

PARAFON* with PREDNISOLONE

provides spasmolytic, anti-inflammatory, and analgesic action

dosage: One to two tablets three or four times a day.
supplied: Tablets, scored, buff colored, bottles of 36. Each tablet contains PARAFLEX® Chlorzoxazone† 125 mg.; TYLENOL® Acetaminophen 300 mg.; and Prednisolone 1.0 mg.
precautions: The precautions and contraindications that apply to all steroids should be kept in mind when prescribing PARAFON WITH PREDNISOLONE.

1. Swanson, J. N.: Canad. M. A. J. *79*:638 (Oct. 15) 1958.

McNEIL

McNEIL LABORATORIES, INC • PHILADELPHIA 32, PA.

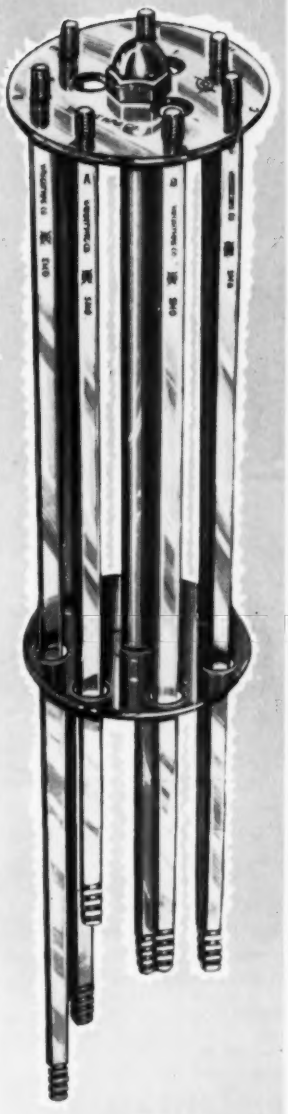
*Trade-mark †U.S. Patent Pending

253439

STREET

SQUARE *Forearm Pins*

SMO STAINLESS STEEL

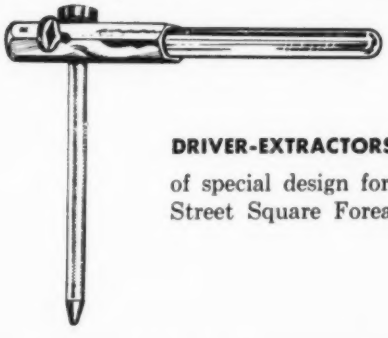


CROSS SECTION

This trade mark is your guarantee of the utmost in quality and workmanship. The Street Square Forearm Pins for Radius or Ulna are SMO stainless steel—spring tempered to a special rockwell hardness. They are available in seven lengths from 8" through 11" in ½" increments. Diameters are 3.1 mm, 4.0 mm, 4.5 mm, with a choice of blunt or self-broaching points. Both styles are threaded one end only.

STAINLESS STEEL REAMER DRILLS *

are especially designed for use with these pins. They are accurately sized for easiest insertion and maximum holding power of each diameter pin.



DRIVER-EXTRACTORS

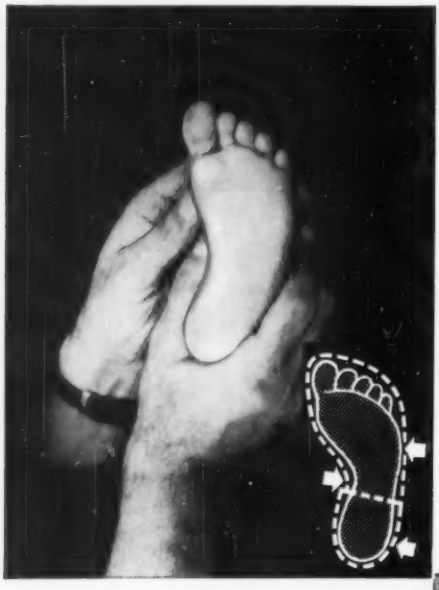
of special design for use with Street Square Forearm Pins.

WRIGHT MANUFACTURING COMPANY

880-882 ADAMS AVENUE
MEMPHIS 3, TENNESSEE

Write for NEW CATALOGUE to Dept. C-1

the action of the TARSO SUPINATOR SHOE



. . . swings the hind-foot inward, which induces adduction of the fore-foot, elevates the longitudinal arch, and levels the ankle joint.

When the hind-foot is held in a position of inward rotation, the talo-scaphoid and cuneiform-scaphoid joints will remain locked under weight bearing. The arch will not collapse and the ankle will not roll inward.

Correction is accomplished by the specially shaped last. Medially wedged and extended heels promote better weight distribution. Medially extended counters are used for utmost control.

BEFORE AFTER

The Tarso Supinator shoe is used successfully in treatment of the plano-valgus foot. Pronation is controlled at once. Permanent improvements in walking and in foot posture will generally occur. Patients find Tarso Supinators notably comfortable.

Tarso Shoes are ready-made and are available on prescription from dealers in most cities. Write for catalog and name of nearest dealer.

T.S. TARSO SUPINATOR REGUS PAT OFF SHOE

FOR FLAT FEET

M. J. MARKELL SHOE COMPANY, INC.

332 SOUTH BROADWAY • YONKERS, NEW YORK

ORIGINATORS OF TARSO PRONATOR® AND TARSO MEDIUS® SHOES

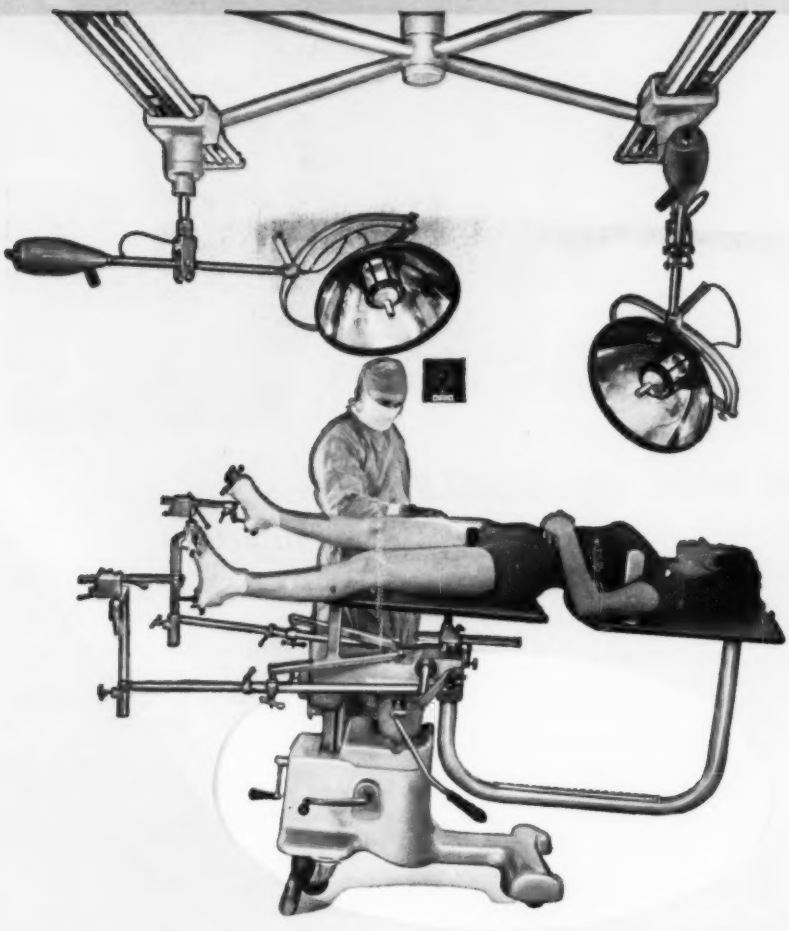
Tarso shoes have been in use for over twenty-five years.

In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

Precise Positioning and Positive Fixation—

with AMERICAN-Albee

ORTHOPAEDIC *and* FRACTURE TABLE



● Acknowledged as the most versatile in its field, the American-Albee Orthopaedic and Fracture Table enables the orthopaedic surgeon to achieve "perfect positioning" of short, tall, obese and even infant patients . . . easily and rapidly. It is designed to accommodate closed fracture reductions as well as the increasing demands of complex orthopaedic surgery. Thus, to orthopaedists the world over, the name American-Albee identifies the table of choice for this comprehensive branch of medicine.

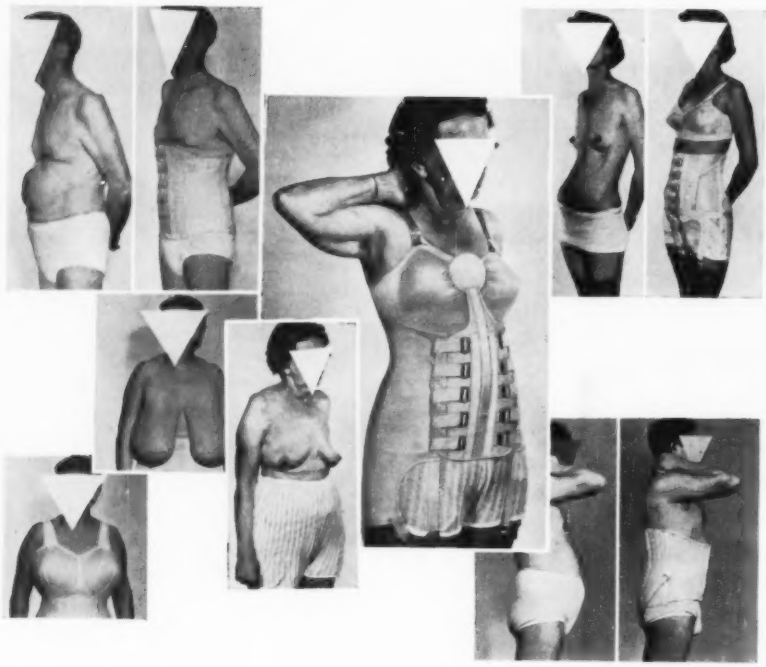
Ideal complement to this improved table is the American DV-22 Major Surgical Light . . . a new concept in easily maneuverable illumination . . . cool, shadowless, color-corrected . . . intensity to 10,000 foot candles *from a dual source.*

● *Write for Fracture Table Brochure TC-100R and Major Light Catalog LC-121R.*

Offices in 14 Principal Cities

AMSCO

AMERICAN STERILIZER



SEE FOR YOURSELF

that no case is too difficult for SPENCER DESIGNERS

However complicated the problem of providing effective, therapeutic support for your patient, the Spencer Corsetiere is trained to follow your instructions carefully. Spencer's patented new Control Support enables her to support and realign the patient's body quickly and comfortably before measuring . . . so that the finished Spencer will realign the body *as it should be*. From the information she submits to Spencer Designers, a Spencer Body or Breast Support is individually designed, cut and made to meet your medical aims with great accuracy.

Spencer's Individual Designing Service is available to you through Corsetieres specially trained to help you help your patients.

SPENCER®

individually designed supports for women, men and children

SPENCER, INCORPORATED
19 Ellsworth Ave., New Haven 7, Conn.
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☐ Send me the name of the nearest Spencer Corsetiere.

☐ I would like an office demonstration of Spencer benefits and features.

Name _____

Address _____

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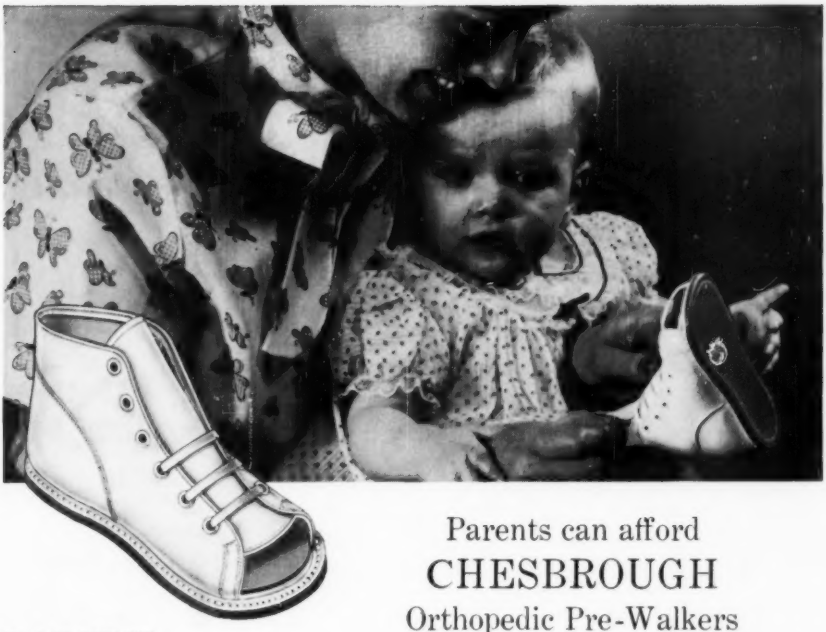
9-59

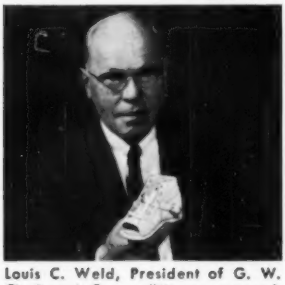


Parents can afford
CHESBROUGH
Orthopedic Pre-Walkers

No. 1400 OPEN TOE
Straight-line symmetrical last, firm heel, no back seam. Adaptable to Denis Browne Splints.

No. 1300 CLOSED TOE
Lace-to-toe design permits snug, gentle fit. Perfectly smooth inside.

No. 1700 CLUBFOOT, OPEN TOE
Special outflare last, sturdy instep strap to stabilize heel.



Louis C. Weld, President of G. W. Chesbrough Co. . . . "My own personal experience led to the development of the new Chesbrough Shoe."

Here are Orthopedic infants' shoes that meet rigid requirements of corrective footwear yet fit the family budget.

High-quality workmanship and fine leathers with orthopedically correct lasts are combined in Chesbrough Shoes to sell at a moderate price.

Any parent whose child requires orthopedic correction knows the expense is great, as frequent purchase of new shoes is required.

This problem was brought home to Chesbrough's president, Louis C. Weld, three years ago, when a child in his own family needed such a shoe. Recognizing the need for orthopedic shoes at an economical price, Chesbrough Company put their 60 years of shoemaking experience to work and Chesbrough Corrective Pre-Walker Shoes were born.

All shoes in unlined white elk, sizes 000 to 4, narrow and wide. Available in full pairs, split pairs or single shoes (no extra charge for half pairs).

MAIL COUPON FOR FREE DESK SAMPLES

G. W. CHESBROUGH CO.
807 Smith Street, Rochester 6, N. Y.

NAME .....

ADDRESS .....

CITY ..... STATE .....

YOUR ORTHOPEDIC SHOE DEALER:

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In answering advertisements, please mention *The Journal of Bone and Joint Surgery*.

OSTIC® gives you something better
in a plaster bandage

THE PROOF IS IN THE ALL-DAY, EVERYDAY USE

You can feel the difference immediately —OSTIC is creamy, gentle, never gritty

Strong, fast and precise, this is the all-around, easy-to-use bandage so popular in teaching centers. It delivers at least 97% of the original plaster to the cast because of the inseparable bonding of plaster and starch-free crinoline. Available in two settings of Fast (5 to 9 min.) and Extra-Fast (2 to 5 min.).

Ostic plaster casts stand up under all kinds of wear. Even with the more unusual fractures, as well as rambunctious little patients. And Ostic is consistent. Every time you open that waterproof package you know you're going to get the same good results you had with the last one.

For strength that meets every need, and creaminess that is kind to your hands all day, rely on OSTIC Plaster Bandages.

Ostic comes in individual airtight packages which prevent pre-setting. Plaster stays fresh indefinitely.

Curity®
OSTIC®
PLASTER BANDAGES

Bauer & Black
DIVISION OF THE KENDALL COMPANY

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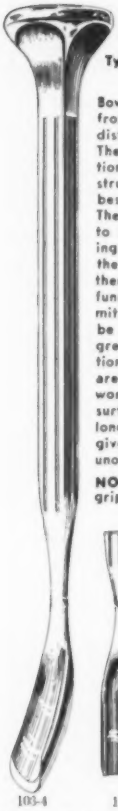
ACCLAIMED BY SURGEONS AS THE LIFETIME INSTRUMENTS

Because of Their Quality and Durability

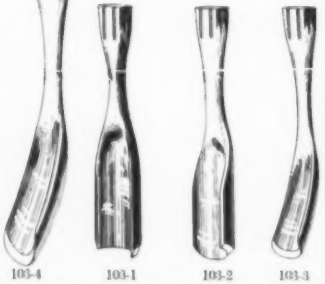
GOUGES



Type 440C Stainless Steel—Swedish Hand Forged

Bowen Gouges, Hand Forged from 44C stainless steel, are of distinctive design and style. The forging method of fabrication produces a fine grain instrument in curves and shapes best desired by the surgeon. The edges are hollow ground to keen razor sharpness resulting in a minimum of effort on the part of the surgeon to make them perform their intended function. The four styles permit a wide range of work to be done. They will be found of great value in bone reconstruction work. The Curved Gouges are particularly adaptable to work in and around contoured surfaces. The fine balance and long square grooved handles give a feeling of sure grip and unobstructed vision.

NOTE: Available with finger grips if desired.



103-4 103-1 103-2 103-3

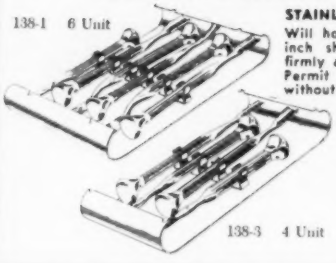
Consult Your Dealer or Write to:

BOWEN & COMPANY, Inc.

P. O. BOX 5818

BETHESDA, MARYLAND

INSTRUMENT TRAYS



138-1 6 Unit

138-3 4 Unit

STAINLESS STEEL

Will hold any instrument with a ⅜-inch shank. Will hold instruments firmly and prevent possible damage. Permit sterilization of instruments without individual handling.

FEATURES:

"Four and six unit trays will nest, one on top of the other." "Trays may be placed on end." "Requires less storage space." "Instruments cannot fall out if tray is turned over."

HATT SPOON—SPINAL GOUGE

103-5

In spinal and arthroplasty surgery, the use of the Spinal Gouge along with our Arthroplasty and Goose Neck Gouges give the surgeon a combination of instruments best suited in this often difficult and intricate work.

FEATURES: "Keen cutting edge." "Hardened and tempered to rigid specifications for maximum quality." "Thumb rest handle designed to insure grip and prevent slipping." "Long handles for unobstructed vision."

No. 103-5 Hatt Spoon—Spinal Gouge.

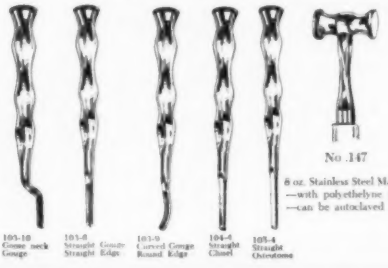
NOTE: Available with finger grips if desired.

CHISELS—GOUGES—OSTEOTOMES

For Surgery of the Hand

Type 440C Stainless Steel—Swedish Hand Forged

Contoured handles to comfortably fit the fingers of the hand so that holding the instruments will be easy and with a secure feeling. The average overall length of the instruments is approximately 6½ inches. The chisels and osteotomes are available in a goose neck blade such as illustrated below by the goose neck gouge.



103-10 Goose neck Gouge | 103-0 Straight Gouge Straight Edge | 103-9 Curved Gouge Round Edge | 104-6 Straight Chisel | 105-4 Straight Osteotome

No. 147

8 oz. Stainless Steel Mallet —with polyethelyne grip —can be autoclaved

you can expect
your patients
with low back pain
to return to
normal activity
when you prescribe

Trancopal

case profile no. 2758

A middle-aged man had intermittent low back pain that he attributed to injuries received in an automobile accident three years previously. The pain radiated down both legs, making the patient walk bent over. He also had difficulty getting out of bed and had to pull his knees up and roll out. Any heavy lifting precipitated a new attack, and he tired easily.

Findings on x-ray of the thoracic and lumbar spine gave negative results. Findings from other laboratory studies were within normal limits. A herniated disc, although still a possibility, was temporarily ruled out by the neurologic examination. Previous treatment consisted of analgesics and steroids (without success), and narcotics were given during severe attacks.

Receiving a dosage of Trancopal, 100 mg., three times a day, this patient is able to walk almost normally and carry on his regular activities as long as he does not overexercise. He has been taking Trancopal over seven months with excellent relief of symptoms. No side effects have occurred. *Clinical Report on file at the Department of Medical Research, Winthrop Laboratories.*

INDICATIONS: Musculoskeletal: Low back pain (lumbago, sacroiliac pain); neck pain (torticollis, etc.); bursitis; rheumatoid arthritis; osteoarthritis; disc syndrome; fibrositis; ankle sprain; tennis elbow; myositis; postoperative muscle spasm.

Psychogenic: Anxiety and tension states; dysmenorrhea; premenstrual tension; asthma; angina pectoris; alcoholism.

Dosage: 100 to 200 mg. orally three or four times daily. Relief of symptoms occurs in fifteen to thirty minutes and lasts from four to six hours.

Supplied: Trancopal Caplets® 100 mg. (peach colored, scored), bottles of 100.

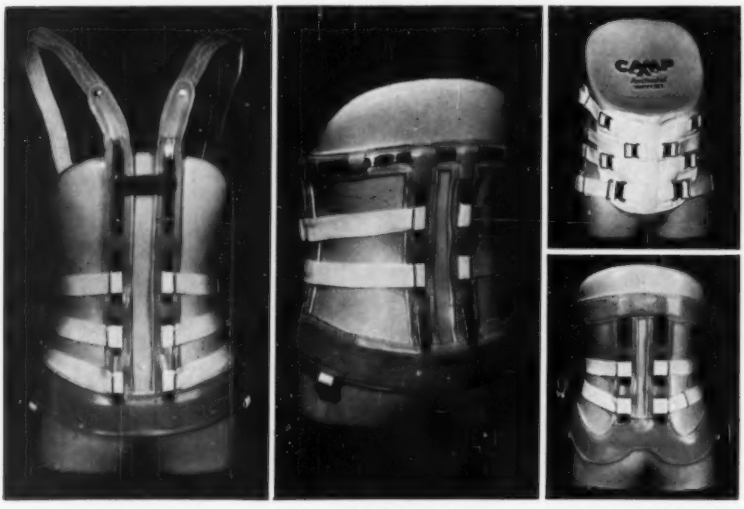
Trancopal®

the first true TRANQUILAXANT

Winthrop LABORATORIES • New York 18, New York

1393M . Trancopal (brand of chlormezanone) and Caplets, trademarks reg. U.S. Pat. Off.

NEW CAMP PREFABRICATED BRACES



TAYLOR BRACE

REGULAR KNIGHT SPINAL BRACE

BUTTERFLY KNIGHT SPINAL BRACE

New, easily shaped, lightweight aluminum braces . . . a Taylor and two types of Knight Spinal . . . a Butterfly and a straight pelvic band model . . . offer effective and limiting movement in the region of the lower lumbar spine. The inside padding is a blanket material covered with soft horsehide. The outside has a protective durable elk leather sheathing. Pelvic bands are enclosed. Uprights, side bars and rib band are partially exposed for shaping except Butterfly type rib band. Pelvic, rib band and shoulder straps are sewn and riveted to braces. Detachable straps attach to back uprights with ferruled links, with slide buckles for attaching to apron fronts. Front pads of satin covered foam rubber have outside white coutil covering with devices for holding truss hooks, hose supporters and/or perineal straps. All three types are available in small, medium and large sizes.

S. H. CAMP and CO.
Jackson, Michigan

CAMP

S. H. CAMP and CO., of Canada, Ltd.
Trenton, Ontario

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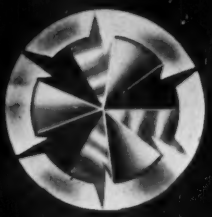
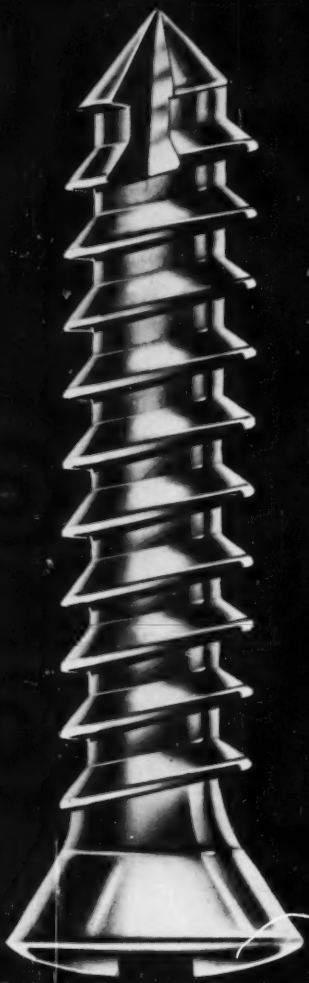
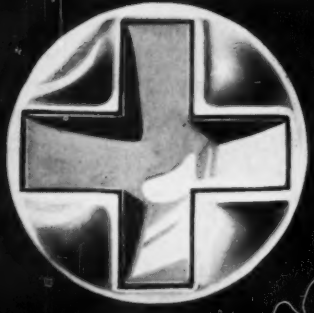


Bechtol Radial Fluted Point



Cruciate Head uses standard screwdriver



NEW *Richards* BONE SCREW

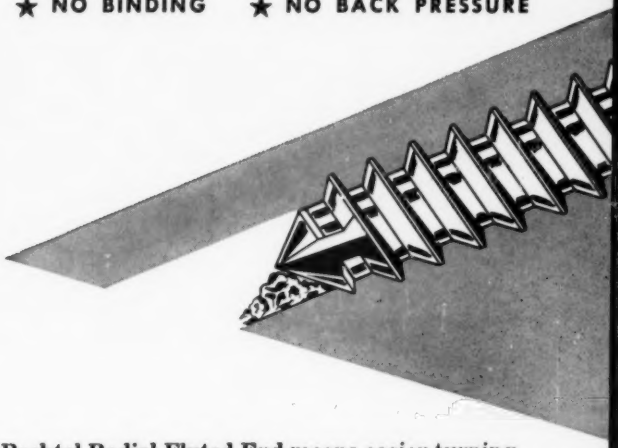
(SMO Stainless)

with Bechtol Radial Fluted Point

and buttress threads

★ TURNS EASIER ★ HOLDS BETTER

★ NO BINDING ★ NO BACK PRESSURE



Bechtol Radial Fluted End means easier turning because flutes move bone crumbs ahead, prevent them from clogging threads and causing screw to bind. Tests prove 50% less torque is required.

Buttress threads increase screw's holding power and eliminate back pressure.

Micrometric accuracy in manufacture assures perfectly true shank for maximum ease of entry and greatest possible thread grip.

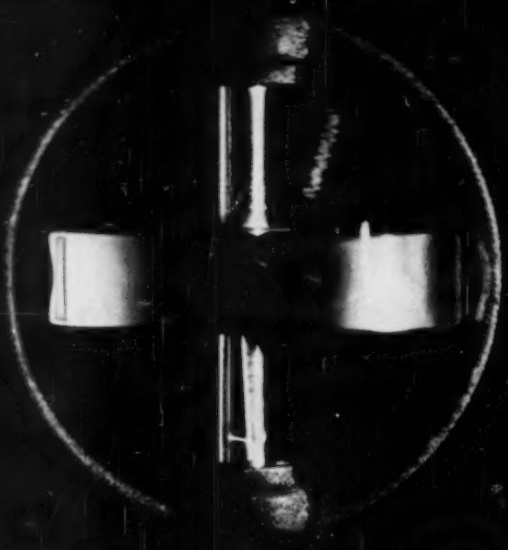
No special instruments needed. Precision-cut Cruciate Head requires standard screwdriver. Regular 7/64" diameter drill bit used. Available in standard bone screw lengths.

Write for information.

Richards **MANUFACTURING COMPANY**

756 Madison Avenue, Memphis 3, Tennessee

minimal disturbance
of the patient's chemical and psychic balance...



still unsurpassed for total corticosteroid benefits

Aris

Substantiated by published reports of leading clinicians:

- **effective control** of allergic and inflammatory symptoms[1-20]

- **minimal disturbance** of the patient's chemical and psychic balance[1,4,5,8-19]

tocort®

Triamcinolone LEDERLE

At anti-inflammatory and antiallergic levels ARISTOCORT means:

- **freedom from salt and water retention**
- **virtual freedom from potassium depletion**
- **negligible calcium depletion**
- **euphoria and depression rare**
- **no voracious appetite — no excessive weight gain**
- **low incidence of peptic ulcer**
- **low incidence of osteoporosis with compression fracture**

Indications: rheumatoid arthritis; arthritis; respiratory allergies; allergic and inflammatory dermatoses; disseminated lupus erythematosus; nephrotic syndrome; lymphomas and leukemias.

Precautions: With ARISTOCORT all traditional precautions to corticosteroid therapy should be observed. Dosage should always be carefully adjusted to the smallest amount which will suppress symptoms. After patients have been on steroids for prolonged periods, discontinuance must be carried out gradually.

Supplied: Scored tablets of 1 mg. (yellow); 2 mg. (pink); 4 mg. (white); 16 mg. (white).

Diacetate Parenteral (for intra-articular and intrasynovial injection). Vials of 5 cc. (25 mg./cc.).

List of References 1-20 supplied on request.

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For the baby with a club foot

EVERY DOCTOR recognizes the absolute necessity for a baby with a club foot wearing just the right shoe. And doctors know there is a Junior Arch Preserver Shoe built on a last designed to meet just this special need.

The Junior Arch Preserver Pre-Walker Club Last is designed to abduct from the heel to the forepart of the foot. The medial side is extended to fully cover and control the first great toe. The strap adjusts to properly seat the heel. And it is available in single shoes for either right or left foot.

Doctors know Junior Arch Preserver Shoes, made by three generations of master craftsmen, are designed with the greatest possible care—and that Medic makes a shoe to fill every doctor's shoe prescription needs.

For the name and address of the store nearest you carrying Junior Arch Preservers, write directly to

Medic Shoe Manufacturers, Inc.

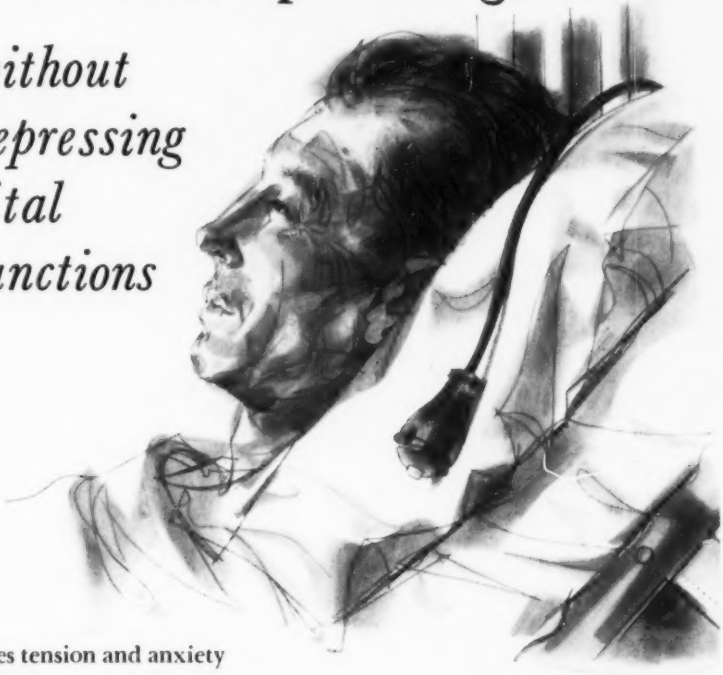
1212 WOOD STREET,
PHILADELPHIA 7, PA.

Junior ARCH PRESERVER Shoes by MEDIC

The complete line of feature shoes for children that doctors recommend

Relieves Pre-op Apprehension
Aids Post-op Management

without depressing vital functions



- relieves tension and anxiety
- helps provide "a good night's sleep"
- facilitates early ambulation
- relaxes skeletal muscle

Miltown has no adverse effects on cardiovascular, respiratory or gastrointestinal functions and causes no depressive "hang-over"

Miltown®
meprobamate (Wallace)

Miltown is available in 400 mg. scored and 200 mg. sugar-coated tablets; bottles of 50.

WALLACE LABORATORIES
New Brunswick, N. J.

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1. Kern, E. C.: Preoperative sedation with meprobamate (Miltown) in surgery of eye, ear, nose and throat. Eye, Ear, Nose & Throat Month. 36:408, July 1957.
2. Lamphier, T. A.: The value of meprobamate preoperatively. Maryland M.J. 7:627, Nov. 1958.
3. Lamphier, T. A.: Meprobamate—an important adjunct to post-operative surgical care. J. Maine M.A. *48*:357, Oct. 1957.
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5. Rushia, E. L.: Preliminary report on the use of meprobamate for pre-anesthetic sedation. J.M.A. Georgia *46*:93, March 1957.
6. Sadove, M. S. and Schiffrin, M. J.: Tranquilizers in surgery, in Surg. Clin. North America, Saunders, Philadelphia, Feb. 1959, pp. 231-244.

CW-8879

NO MORE

because VITAGENIC

THE PRESCRIPTION-BUILT **EXTRA-FIRM MATTRESS**

CURLED HAIR FOR RESILIENT FIRMNESS
Firm body support without undue pressure on nerve points or interference with circulation.

HAND-STITCHED SAG-FREE SIDEWALLS
Sag-free edges allow patients with back injuries to get in and out of bed with less effort and greater stability. 12% more usable sleeping area.

NEEDS NO "MAKESHIFT" INTERPOSED BOARD

Physicians and orthopaedic specialists know that a bedboard—even one that is interposed in a mattress—is only an attempt to correct a structural weakness and inadequacy in the mattress itself. The VITAGENIC by AIRE-LOOM requires no "make-shift" additions.

The VITAGENIC is a mattress and box spring combination made according to the exact specifications of registered orthopaedic specialists. It is an important—almost essential—adjunct to your plan of treatment for pain of backache or sacroiliac injury. You prescribe the exact degree of firmness necessary for your patient as well as any other features required for treatment or comfort.

THE VITAGENIC IS GUARANTEED FOR 15 YEARS.

Aireloom

For full details on the prescription-built VITAGENIC, contact:

BEDDING CO. • 1119 Westminster Ave., Alhambra, Calif.

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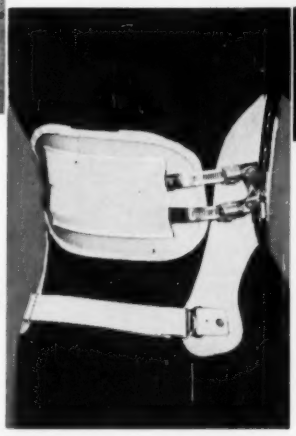

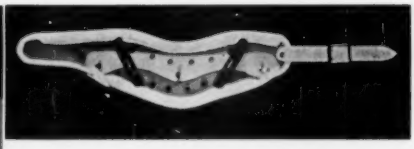

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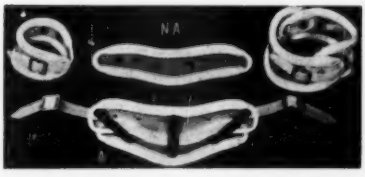

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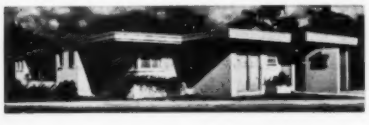

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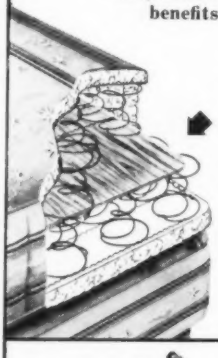

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WOLINSKY, E., SMITH, M. M., and STEENKEN, WM. JR.: Tuberculocidal Activity of Clorpactin, A New Chlorine Compound, Antibiotic Medicine, 1: 382–384, July 1955.

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GLIEDMAN, M., GRANT, R., VESTAL, B., ROGERS, C., KARLSON, K. E.: Clorpactin, A Surgical Adjunct: Antimicrobial and Tumorcidal Properties. Presented at the Surgical Forum of the American College of Surgeons, Oct. 17, 1957.

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VOL. 41-A, NO. 6 SEPTEMBER 1959

The Journal of
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American Volume

Treatment of Malignant Tumors of the Extremities by Perfusion with Chemotherapeutic Agents*†

BY EDWARD T. KREMENTZ, M.D., OSCAR CREECH, JR., M.D., ROBERT F. RYAN, M.D.,‡ AND JACK WICKSTROM, M.D., NEW ORLEANS, LOUISIANA

From the Department of Surgery and Division of Orthopaedics, Tulane University School of Medicine, the Tulane Surgical Service at Charity Hospital of Louisiana at New Orleans, Touro Infirmary, and the United States Public Health Service Hospital, New Orleans

In order to increase salvage and to obtain better palliation in the treatment of malignant disease, recent emphasis has been directed toward the use of chemotherapeutic agents. In general, the cancerocidal drugs have been used as adjuvants to surgical excision or to irradiation in the treatment of curable lesions or for palliation in extensive regional or disseminated malignant conditions. The limiting factor to the use of chemotherapy, however, has been the toxic effects on the bone marrow and on the gastro-intestinal tract. In an attempt to limit these systemic toxic effects and to obtain the benefit of increased dosage a method has been developed in our laboratory whereby the tumor-bearing area could be temporarily isolated and perfused by utilizing a pump-oxygenator[9]. This report is concerned with our experiences with this method in the treatment of malignant tumors of the extremities.

MATERIALS AND METHODS

Methods were developed in the laboratory for the isolation and perfusion of the extremities, mid-gut, and liver in the experimental animal[9,10]. These procedures have also been applied clinically for perfusion of the extremities, lung, breast, pelvis, oropharynx, and total body[2,7,8,10]. To date seventy-three cases have been treated by this method[3]. Among these there were seventeen patients with malignant melanomata, seventeen with sarcomata, and three with carcinomata involving the extremities. Perfusion of the lower extremity is accomplished through the external iliac, the common femoral, or the superficial femoral vessels, depending upon the location of the tumor. In the upper extremity, the subclavian, axillary, or brachial vessels are used. The technique for the isolation and perfusion of the lower extremity is as follows. With the patient under spinal or general anesthesia, the common femoral artery and vein are exposed through a

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 26, 1959.

† This work was supported in part by the Anna Fuller Fund, the National Cancer Institute Health Service Grant-in-Aid CY-3782-CY, and the American Cancer Society Institutional Grant IN-24A.

‡ Clinical Fellow, American Cancer Society.

transverse incision below and paralleling the inguinal ligament. The vessels are isolated and secured with umbilical tapes placed about three centimeters apart on both artery and vein. Heparin is administered intravenously in a dose of two milligrams per kilogram of body weight. The vessels are then occluded by the tapes and a longitudinal incision approximately 0.5 centimeter in length is made in the artery between the tapes (Fig. 1). A No. 16 or No. 18 polyethylene catheter is inserted distally in the femoral artery for a distance of approximately five centimeters, and the distal tape is tightened about it. The femoral vein is cannulated in a similar manner. The catheters are then attached to the extracorporeal circuit consisting of two TM-4 sigmamotor pumps and a bubble-dispersion oxygenator (Fig. 2). The latter is a disposable plastic bag with a reservoir capacity of 500 cubic centimeters and an oxygenating capacity of 1500 to 2000 milliliters per minute. The circuit is primed with 500 cubic centimeters of fresh citrated

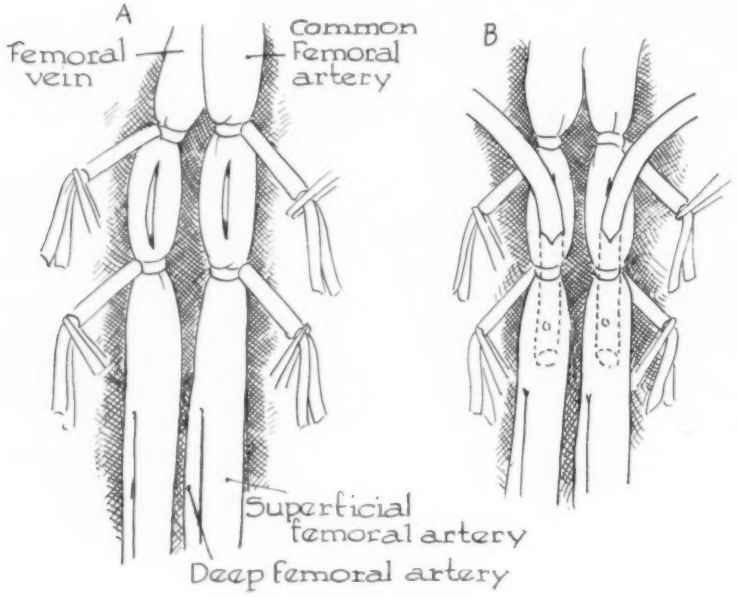

FIG. 1

Diagram showing method of isolation of common femoral artery and vein, incisions in vessels, and position of polyethylene catheters after insertion. The tapes used consisted of a loop of umbilical tape pulled through a four-centimeter section of a No. 18 French rubber catheter.

blood to which twenty milligrams of heparin have been added just prior to use. A tourniquet, passing through the gluteal fold and over the iliac crest, is drawn tightly around the extremity proximal to the point of insertion of the catheters (Fig. 3). Either a one-half inch latex tube or several lengths of an Esmarch bandage are satisfactory as a tourniquet.

After the perfusion is started a balanced flow is obtained within a few minutes. A high degree of oxygen saturation is indicated when a slight color differential is visible between the blood in the arterial and in the venous lines. The chemotherapeutic agent is then added to the system just proximal to the arterial pump-head to prevent streaming and to allow adequate mixing. Streaming has been observed when Evan's blue dye has been injected distal to the arterial pump-head in perfusion of certain regions such as the mid-gut. With this situation, the dye was seen in some, but not in all, of the arcades of the treated bowel, indicat-

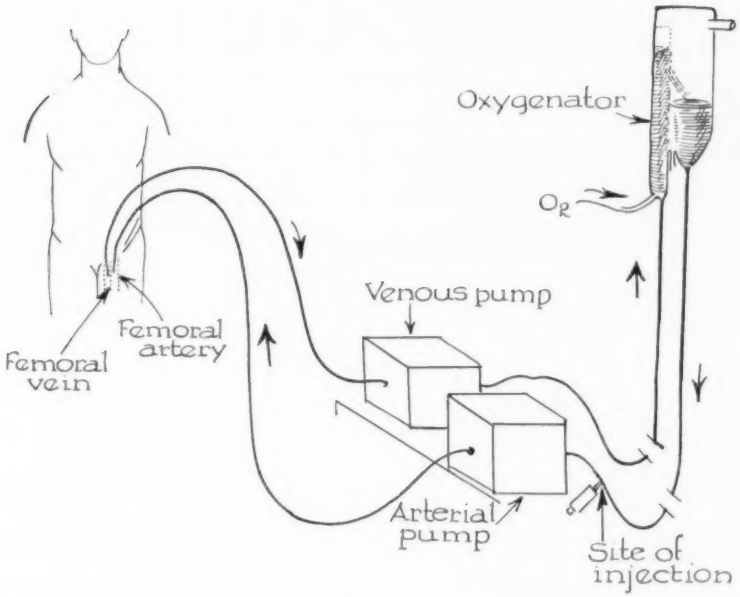

FIG. 2

Diagram showing patient undergoing femoral artery perfusion, sigmamotor pumps, oxygenator, and drug injection site.

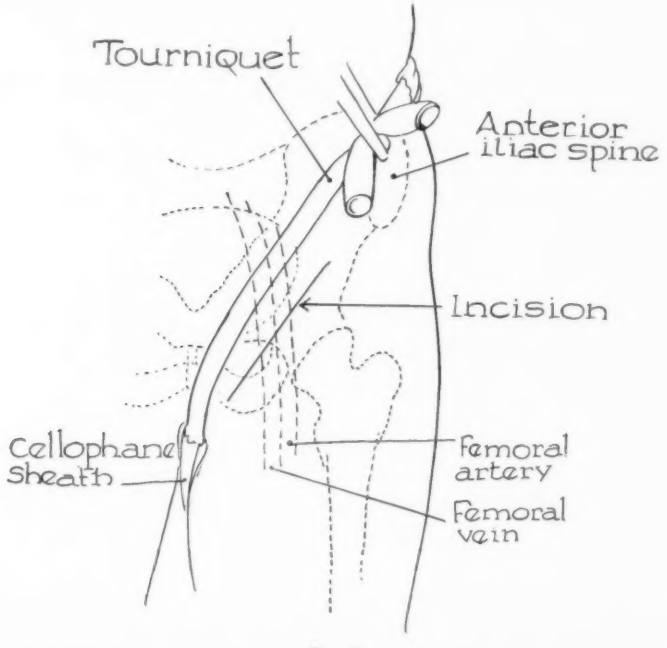

FIG. 3

Diagram showing incision for common femoral artery perfusion and site of application of tourniquet. Sterile tourniquet encased in cellophane sheath runs from groin to iliac crest. When tightened, portion in field remains sterile.

ing incomplete mixing. Rapidly acting agents are added in four aliquots at five-minute intervals while the longer acting drugs are administered in two doses at ten-minute intervals. In the latter instance, perfusion is usually continued for thirty minutes after administration of the second dose. Upon completion of the perfusion, particularly if long-acting drugs have been used or if there has been an accumulation of toxic end products, an attempt is made to remove the perfusate. The arterial pump is shut off and the venous line is allowed to drain 500 to 800 cubic centimeters of the perfusate. This may be further facilitated if new blood or dextran is pumped into the arterial circuit. The tourniquet is then released, the catheters are removed, and the artery and vein are repaired with running sutures of 00000 arterial silk. Hexadimethrine bromide, in the amount equal to that of heparin given, is administered intravenously at the time of the repair of the femoral vessels to counteract the anticoagulant effects of heparin.

TABLE I

DOSAGE FOR COMMON FEMORAL ARTERY ISOLATED PERFUSION

Nitrogen mustard	0.8	milligram per kilogram of body weight
Phenylalanine mustard	1.5	milligrams per kilogram of body weight
Actinomycin D	.05	milligram per kilogram of body weight
Triethylene thiophosphoramide	0.8	milligram per kilogram of body weight
5-fluorouracil	20	milligrams per kilogram of body weight

The cancerocidal agents * employed in this series were as follows: nitrogen mustard (HN_2), phenylalanine mustard, triethylene thiophosphoramide, actinomycin D, and 5-fluorouracil. Nitrogen mustard, actinomycin D, and 5-fluorouracil are relatively short acting while the rest have longer periods of action. Several other drugs, including triethylene melamine and chlorambucil, are presently under study.

The dosages recommended for perfusion of the common femoral artery are listed in Table I. Several factors are of importance in determining the maximum amount of an agent that can be given by this technique. The lower extremity, perfused through the common femoral artery, will tolerate the usually recommended systemic dose. This represents a six to eightfold increase in concentration of the drug since the lower extremity constitutes one-sixth or less of the total body mass. Animal experiments suggest that the amounts of chemotherapeutic agents may be increased beyond the limits described above and in recent clinical experience this has been done without any apparent increase in toxic effects [2,11]. If the patient is obese or has had previous chemotherapy or irradiation the dosage should be lowered. Also, if perfusion is to be confined to a smaller area than that supplied by the common femoral artery the dosage should be reduced. Thus, while 0.8 milligram per kilogram of body weight of nitrogen mustard would be used for perfusion of the common femoral artery the dose would be reduced to 0.4 or 0.6 milligram per kilogram of body weight for perfusion of the axillary artery.

RESULTS

There have been forty-five perfusions performed in thirty-seven patients, including seventeen with malignant melanoma, seventeen with some type of sarcoma, and three with a primary or metastatic carcinoma of the extremity.

It is as yet impossible to evaluate the results of treatment in these patients

* Drug acknowledgments: nitrogen mustard, Merck Sharp & Dohme through the courtesy of James M. Sherwood, M.D.; phenylalanine mustard, M. D. Anderson Hospital through the courtesy of A. Clark Griffin, M.D.; phenylalanine mustard and chlorambucil, Burroughs Wellcome & Co. through the courtesy of Donald S. Searle, M.D.; actinomycin D, triethylene thiophosphoramide, and triethylene melamine, Lederle Laboratories through the courtesy of James M. Ruegsegger, M.D.; 5-fluorouracil, Hoffman-La Roche Inc. through the courtesy of Thomas V. Morton, Jr., M.D.

with malignant disease because of the short time that the study has been in progress. An attempt has been made, however, to determine the present status of each patient but it must be kept in mind that their status is constantly changing. Some of the patients have been perfused as an adjuvant to the usual surgical procedures, although most have been treated for control or palliation of extensive but regionally confined disease. In the latter group are patients who have refused further excisional surgery or those whose cancers are so biologically active that further radical surgery is not justified. In recording the results, patients have been

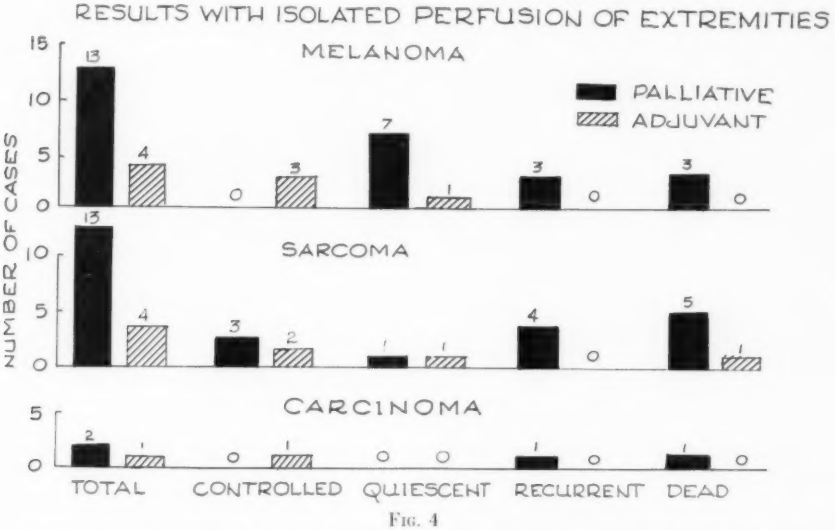

FIG. 4

Results of treatment with isolated perfusion of the extremities in patients with melanoma, sarcoma, and carcinoma.

categorized as dead, or alive with disease that is recurrent, quiescent, or controlled. These categories are defined as follows: *dead*, all patients who have died with active malignant disease; *recurrent*, those patients with lesions which may or may not have regressed after perfusion but who now have a recurrence; *quiescent*, those patients in whom the disease, while still apparent in the extremity, is inactive or quiescent; *controlled*, patients in whom there is no clinical evidence of residual disease (Fig. 4).

Among the seventeen patients with malignant melanoma, fourteen had disease confined to the lower extremities, and three to the upper extremity. Of thirteen patients treated for palliation, three are dead. Two of these had the local tumor controlled by perfusion but subsequently died of distant metastases. The third patient died as a result of an excessive dose of phenylalanine mustard with inadequate isolation of the extremity. This patient died with a marked leukopenia and with severe toxic effects thought to result from massive destruction of the tumor. Among the ten living patients treated for palliation, three have recurrent disease and seven are considered quiescent. It must be emphasized that this group is composed of patients with far advanced but regionally confined disease which, if treated conventionally, would require radical amputation (Figs. 5-A and 5-B). Four patients with melanoma had perfusion as an adjunct to surgical excision. In three of these the disease is controlled and in one there was evidence of local recurrence with the appearance of a satellite nodule outside the grafted area; this was excised and no further activity has been noted. Of the patients with melanoma two have been perfused on three occasions and one perfused twice. These patients were perfused with phenylalanine mustard alone except for two perfusions with

TABLE II

Patient	Dates of Perfusion	Site	Drug	Chemotherapy	Surgery Following Perfusion	Leukocyte Depression: Comparison with Pre-perfusion White Blood Count (*First Perfusion*)	Results as of May 15, 1959
J.S.P.	6/11/58	Foot	Phenylalanine mustard	Palliative	Biopsy	No change	Quiescent
M.W.	1/7/58	Heel	Phenylalanine mustard	Adjuvant	Groin dissection	20 to 50%	Controlled
H.S.	1/10/58 4/22/58 12/12/58	Foot	Phenylalanine mustard (×2) Phenylalanine mustard and triethylene thiophosphoramide (×1)	Palliative	Multiple excisions	No change	Local disease quiescent; minimal change in pre-perfusion pulmonary metastases
J.K.	2/21/58	Leg	Phenylalanine mustard	Palliative		75 to 100%	Died 16 days post-perfusion; overdosage of drug with poor isolation
E.H.	2/28/58	Calf	Phenylalanine mustard	Palliative	Groin dissection	20 to 50%	Local disease quiescent; death from metastases, 9/4/58
L.G.	4/8/58 7/28/58 11/17/58	Foot	Phenylalanine mustard (×2) Triethylene thiophosphoramide (×1)	Palliative	Biopsy	75 to 100%	Recurrent, to be re-treated
K.S.	5/5/58 9/22/58	Thigh	Phenylalanine mustard (×2)	Palliative	Multiple excisions and node dissection	50 to 75%	Quiescent
F.W.	5/6/58	Heel	Phenylalanine mustard	Adjuvant	Excision	50 to 75%	Recurrent satellite lesion; excised; now quiescent
R.W.	7/10/58	Upper arm	Phenylalanine mustard	Adjuvant	Primary excision and node dissection	50 to 75%	Controlled
L.K.	7/18/58	Heel	Phenylalanine mustard	Palliative		20 to 50%	Quiescent
E.S.	7/26/58	Thumb	Phenylalanine mustard	Adjuvant	Amputation	No change	Controlled
E.F.	9/18/58	Heel	Phenylalanine mustard	Palliative	Excision	75 to 100%	Quiescent
C.C.	9/26/58	Thumb	Phenylalanine mustard	Palliative	Amputation	75 to 100%	Local disease controlled; pulmonary metastases
R.M.	9/26/58	Calf	Phenylalanine mustard	Palliative		50 to 75%	Quiescent
A.B.	8/13/58	Arm	Phenylalanine mustard	Palliative	Excision of axillary mass	75 to 100%	Dead
B.M.	10/14/58	Leg	Phenylalanine mustard	Palliative	Groin dissection	0 to 20%	Quiescent
M.A.	10/23/58	Leg	Phenylalanine mustard	Palliative		No change	Quiescent

TABLE III

Patient	Dates of Perfusion	Type	Site	Drug	Treatment: Chemotherapy	Treatment: Surgery	Leukocyte Depression: Comparison with Preperfusion White Blood Count	Results as of May 15, 1959
B.P.	10/4/57	Rhabdomyosarcoma	Forearm	Nitrogen mustard	Palliative		50 to 75%	Complete regression followed by metastases, local recurrence, and death, 5/24/58
M.A.	10/15/57	Osteogenic sarcoma	Tibia	Nitrogen mustard	Adjuvant	Amputation	No change	Dead with pulmonary metastases, 1/15/58
E.R.	11/7/57	Liposarcoma	Thigh	Nitrogen mustard	Palliative	Excision	No change	Controlled
H.E.	11/14/57	Alveolar-cell sarcoma	Gastrocnemius	Nitrogen mustard	Adjuvant		No change	Controlled
H.D.	6/3/58	Liposarcoma	Thigh	Nitrogen mustard	Palliative		No change	Dead with generalized metastases, 7/15/58
L.O.	6/30/58	Kaposi's sarcoma	Leg	Nitrogen mustard	Palliative		20 to 50%	Recurrent disease in leg
L.G.	9/26/57	Synovial sarcoma	Hand	Nitrogen mustard	Palliative	Amputation	75 to 100%	Alive with pulmonary metastases
W.P.	10/31/57	Rhabdomyosarcoma	Shoulder	Nitrogen mustard	Palliative		No change	Marked local regression; dead of metastatic disease, January 1958
P.M.	1/30/58 5/8/58 12/1/58	Chondrosarcoma	Forearm	Nitrogen mustard (×1) and phenylalanine mustard (×2)	Palliative	Excision	75 to 100%	Recurrent and retreated, May 1959
M.W.	2/25/58	Myxosarcoma	Leg	Phenylalanine mustard	Palliative	Excision	50 to 75%	Controlled
R.N.	5/16/58 11/25/58	Rhabdomyosarcoma	Thigh	Phenylalanine mustard (×1) and triethylene thiophosphoramide (×1)	Adjuvant	Excision	No change	Quiescent
E.L.	5/22/58	Liposarcoma	Upper Arm	Phenylalanine mustard	Palliative	Excision	75 to 100%	Controlled
M.B.	6/20/58	Fibrosarcoma	Forearm	Phenylalanine mustard	Adjuvant	Amputation	No change	Controlled
V.B.	2/20/58	Osteogenic sarcoma	Humerus	Phenylalanine mustard	Palliative	Amputation	75 to 100%	Converted to "operable"; dead from pulmonary metastases, 7/13/58
O.G.	5/8/58	Periosteal sarcoma	Leg	Actinomycin D	Palliative	Excision	No change	Local regression followed by recurrence
R.H.	10/22/58	Osteogenic sarcoma	Femur	5-fluorouracil	Palliative		No change	Slight improvement; dead from metastases, 12/27/58
R.D.	12/30/58	Fibrosarcoma	Foot	Phenylalanine mustard and triethylene thiophosphoramide	Palliative		20 to 50%	Quiescent

FIG. 5-A

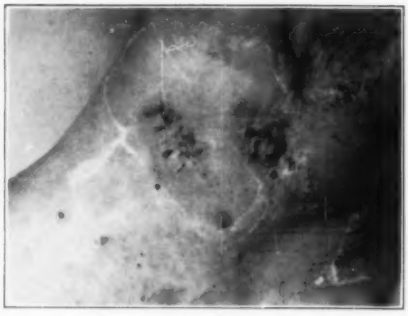

FIG. 5-B

G. B., a woman, sixty-four years old, with malignant melanoma originally excised six years prior to admission. Patient noted onset of multiple dermal metastases one year ago. Re-excision was carried out followed by recurrence (Fig. 5-A). Triethylene thiophosphoramide was given intra-arterially without benefit. Perfusion with phenylalanine mustard resulted in regression of metastases (Fig. 5-B).

FIG. 6-A

FIG. 6-B

M. W., a woman, fifty-four years old, with myxosarcoma of left leg which had been resected fourteen years prior to admission. Recurrence noted two years ago but treatment refused. On admission the lesion was not locally resectable and amputation was refused. Patient was perfused with phenylalanine mustard (Fig. 6-A). Leg twenty-two days after perfusion showed considerable regression of the tumor (Fig. 6-B). Local excision was carried out several days later. Patient showed no evidence of recurrence one year afterward.

a combination of phenylalanine mustard and triethylene thiophosphoramide and one perfusion with triethylene thiophosphoramide. The data on the patients with melanoma are summarized in Table II.

Twenty perfusions have been carried out in seventeen patients with sarcoma of the extremities. In ten the lesions were confined to the lower extremity while in seven the upper extremity was involved. The chemotherapeutic agent was nitrogen mustard in ten perfusions; phenylalanine mustard in six; both phenylalanine mustard and triethylene thiophosphoramide in two, and actinomycin D and 5-fluorouracil in one perfusion each. In these seventeen patients the histological

FIG. 7-A

FIG. 7-B

M. H., a colored woman, fifty years old, with epidermoid carcinoma of thumb of one year's duration (Fig. 7-A). Patient was perfused through the brachial artery with nitrogen mustard. Picture of thumb (Fig. 7-B) eight days later. Amputation carried out two days thereafter. There was no evidence of recurrence one year later.

diagnosis, the site of the lesion, the date of treatment, the therapeutic agent employed, the type of treatment, the leukocyte depression as compared with the pretreatment leukocyte count and the results are recorded in Table III. Of the four patients in whom perfusion was used as an adjunct to surgical excision, one is dead, in another the lesion recurred and is quiescent after retreatment, and in two the disease appears to be controlled. Of the thirteen treated for palliation, five are dead. Two of these had rhabdomyosarcoma which showed significant regression after perfusion but distant metastases later developed and the patients died. Three with osteogenic sarcoma died with pulmonary or disseminated metastases. Among the living patients four have had varying degrees of regression of their tumors with subsequent recurrence while in one the lesion is quiescent and in three the lesions are controlled (Figs. 6-A and 6-B).

Relatively few patients with carcinoma of the extremities have been treated by this method. In a patient with an epidermoid carcinoma of the thumb perfusion was carried out two weeks prior to amputation of the thumb. In the interval partial regression of the tumor occurred (Figs. 7-A and 7-B). Axillary dissection, which is usually combined with amputation in such cases, was deferred in this instance. The patient has had no evidence of recurrence as of May 1959, approximately fourteen months after treatment. Two patients were treated for metastatic adenocarcinoma of the extremities. One was a thirteen-year-old girl with multiple bony metastases of all extremities from an unknown primary mucinous adenocarcinoma. This patient's left lower extremity was perfused with actinomycin D without oxygen. Our plan was to carry out a second perfusion of the right lower extremity at a later date with oxygen to see if a different response could be obtained. While some regression occurred in the perfused extremity, the patient's family refused further procedures, and the patient was discharged. The patient died on December 12, 1958, two months after original treatment. The other patient was a woman, fifty-three years old, with metastatic adenocarcinoma in the tibia from the kidney. There has been some evidence of local improvement in the latter case, although it is too early to evaluate the results.

Local complications in the extremities have been relatively minor. There has been no evidence of arterial or venous thrombosis and edema of the extremity has not been a serious problem. Two patients with malignant melanoma had moderate edema, and one, on whom a groin dissection was performed immediately after perfusion, had rather severe edema. This patient subsequently had a wound infection with partial necrosis of the skin flaps. Among the patients with sarcoma, three had moderate edema and one had severe edema. In one patient (M.H., Figs. 7-A and 7-B) following perfusion of an upper extremity some atrophy of the muscles of the forearm and hand developed which may have been due to an excessive amount of nitrogen mustard.

When the dosage has been high, intense erythema of the skin has occurred followed by brawny edema, similar to that observed following x-ray therapy. This is to be expected since the alkylating agents exert effects similar to those of ionizing radiation. The administration of cortisone seems to speed resolution of this process.

The systemic complications have been minimal following isolated perfusion of the extremities. The amount of nausea or vomiting immediately following the operative procedure has been no greater than that seen after general anesthesia. Depression of bone marrow has been a problem in some cases; however, if isolation is adequate the depression has been minimal. The bone-marrow depression has been followed by study of the peripheral blood counts. Usually all elements are depressed, but the white blood count is the easiest and most reliable to follow. In the thirty-seven patients who had perfusions of the isolated extremities the leukocyte counts were depressed after perfusion 75 to 100 per cent of the original pre-perfusion count in six, 50 to 75 per cent in nine, 25 to 50 per cent in eight, and from 0 to 25 per cent in fourteen. In general, depression of thrombocytes paralleled that of the leukocytes but there has been no gross bleeding and only transient purpura has been observed to result from the thrombocytopenia.

DISCUSSION

It should be emphasized that the tumors included in this series do not respond ordinarily to conventional methods of chemotherapy or irradiation. Thus, it can be assumed that the response observed in many of these patients was the result of the isolation-perfusion technique.

The intra-arterial administration of a chemotherapeutic agent increases its effectiveness as demonstrated by Klopp and associates and by Sullivan. The technique described here includes intra-arterial administration of the chemotherapeutic agents as well as continuous re-circulation of the drug-containing perfusate. Thus, the agent is in continuous contact with the tumor for the duration of perfusion.

The concentration of the chemotherapeutic agent is increased over that obtained by conventional methods since a larger total dose can be given, and the drug is confined to a restricted vascular bed. This results in a six to tenfold increase in drug level. In spite of the large dosages employed, only minimal injury has been observed in perfused normal tissues.

The importance of oxygenating the perfusate is not clearly established. The work of Churchill-Davidson and of Gray has demonstrated that better therapeutic responses are obtained when patients undergoing x-ray therapy have elevated tissue oxygen tension. Most of the chemotherapeutic drugs used in this study exert effects similar to those of ionizing radiation [5] so it may be anticipated that the high oxygen tension would potentiate the cancerocidal action. We have demonstrated experimentally that the toxicity of 5-fluorouracil has been significantly increased by oxygenation [11] and the clinical responses observed tend to confirm this.

Another factor of importance is that the action of the chemotherapeutic drugs is confined to the isolated part so that interference with the host's defense mecha-

nisms is avoided. Furthermore, by washing out the system at the completion of perfusion, toxic end products are largely removed and their absorption minimized.

At present an attempt is being made to improve the results by using combinations of drugs rather than a single agent. This approach has been demonstrated to be effective by Shapiro and his associates and it is hoped by selecting drugs with different sites of action, better results may be obtained. Attempts are also being made to improve the selection of more specific chemotherapeutic agents and thus far we have only clinical observations as a guide. Studies are now underway to develop a sensitivity test. Suspensions of tumor cells are prepared in the patient's own serum and incubated with a chemotherapeutic agent in the same concentration as would be present during perfusion. After an appropriate time the cells are stained with vital stains and examined for viability. The dead cells take up eosin and can be differentiated from the live cancer cells which do not stain. Thus the agent causing the greatest number of dead cells is selected for use. This test is subject to many errors and as yet we cannot rely on this method to select the drug of choice.

Although there are many possible uses for the techniques of perfusion, our principal concern now is the evaluation of highly toxic agents whose therapeutic usefulness is limited by their toxicity. It should be possible to use this method to determine whether new chemotherapeutic agents warrant further development as indicated by their effects on human tumors.

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Transchondral Fractures (Osteochondritis Dissecans) of the Talus*

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Alexander Monro [52] has been credited, possibly erroneously †, with being the first to report the finding of loose osteocartilaginous bodies in the ankle joint. He attributed the condition to trauma but did not classify it as a fracture. Stevenson [73] used the same term in describing a case which he reported in 1925.

In 1888 König [43] observed loose bodies in joints other than the ankle. He referred to them as corpora mobile and speculated about their etiology. He decided that they could not have been caused by any known disease, tumor, or trauma, and concluded, without substantiating evidence, that they must have been produced by a process of spontaneous necrosis. For this hypothetical mechanism he coined the term *osteochondritis dissecans.*

The first use of the term *osteochondritis dissecans* as applied to the ankle joint was made by Kappis [41] in 1922. After that time the names formerly used —loose bodies, joint mice, and corpora mobile—were used as secondary terms. Osteochondritis dissecans became the accepted name and has been used by the majority of authors since 1922.

In 1932 Rendu [60] reported a case of intra-articular fragmentary fracture of the talus. The lesion, as shown by his roentgenograms and as described in his operative note, was the same type that had been reported by Monro and Kappis. Rendu's term has since been used by a few authors.

Since 1932 two separate series of reports of osteocartilaginous lesions of the dome of the talus were published. In one report the lesions were called fractures, in the other they were labeled osteochondritis dissecans. When the case histories and roentgenograms in these reports were studied carefully, it became evident that the authors of the two sets of reports were describing the same condition. In fact, in twenty-eight of the cases reported the authors used the two terms interchangeably.

Because the condition rarely occurs and has been reported under many diagnostic terms, misconceptions and misunderstandings of the lesion have developed. These erroneous ideas have obscured the characteristics of the condition that are significant to the patient, the attending surgeon, and the organizations involved in compensatory payments.

This report is intended to draw attention to the points of misunderstanding and to clarify them in the light of the information gained from a thorough study.

PRESENT SERIES

The following case histories are included for two reasons: to emphasize certain characteristics of the condition, and to illustrate the present-day differences of opinion among surgeons concerning the nomenclature, etiology, and treatment of the lesions.

* Abridgment of the thesis submitted to the Faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfillment of the requirements for the degree of Master of Medical Science for graduate work in Orthopaedic Surgery.

† Monro's original report was not available to us. That report was cited by Barth [8], who said that Monro's lesion was located in the knee, and by Phemister [56], who said it was in the ankle.

FIG. 1

Case 1. Roentgenograms of the ankle five hours after injury showing avulsion fracture from the lateral border of the talar dome with detached chip lying upside down in its bed.

CASE 1. R.E.B., a male railroad worker, thirty years old, had no history of ankle disability until June 7, 1949, when, descending from a locomotive, he stepped down with his left foot on an air hose and turned his ankle. There was a snapping sound in the ankle, accompanied by pain anterior to the lateral malleolus. He was able to walk, but with a limp, and reported that he had sprained his ankle. The ankle showed the characteristics of an inversion sprain: tenderness about the malleolus, increased pain on passive inversion, soft-tissue swelling, ecchymosis, and limitation of motion. Roentgenographic examination on the same day showed a chip fracture of the lateral border of the dome of the talus, the chip lying upside down in the bed from which it had been detached. The other bone components of the ankle joint appeared to be normal (Fig. 1).

An anterolateral incision was made at operation on June 9, 1949. The anterior and middle fasciculi of the lateral collateral ligament were found to be completely disrupted; but the posterior fasciculus was intact, as were the deltoid and interosseous ligaments. The joint was distended with partly clotted blood. Plantar flexion of the foot brought the anterior portion of the fracture into view beneath the anterior lip of the tibia. The inverted loose fragment was extracted; it was a single, intact piece, eighteen by thirteen by five millimeters. One surface was covered with normal articular cartilage; the opposite surface was fresh trabecular bone. The crater from which the fragment had been removed also showed a surface of raw, bleeding bone. The lesion was clearly a new fracture. Since the fragment constituted about one-third of the articular surface of the talar dome, it was deemed advisable to replace it rather than discard it. (The chip could be removed later if lack of blood supply should prevent union.) The chip was inserted in its proper position and impacted, as well as the limited access permitted, into the main fragment of the talus. A toe-to-groin cast was then applied.

Roentgenograms made three weeks later (Fig. 2-A) showed no change from the immediate postoperative films. The cast was replaced by a posterior metal splint for an additional five weeks at which time graduated weight-bearing was begun.

Roentgenograms made at fourteen and twenty weeks with the ankle in plantar flexion showed what appeared to be progressive healing of the fracture with obliteration of the fracture line (Figs. 2-B and 2-C). Clinical improvement was also noted.

During the ninth month, however, with no further injury, there was an increase in pain on walking. Roentgenograms were made with the ankle in dorsiflexion and in plantar flexion. In the plantar-flexion view, the fracture line appeared to be healed, but in dorsiflexion, the fracture line was clearly seen. Its appearance (Fig. 2-D) was almost identical with that seen on the roentgenogram made immediately after reduction. Lateral views showed the fracture line to be visible, but indistinct. Operative removal of the fragment was suggested, but declined by the patient.

Five years after operation the patient returned for examination. He had had no further treatment for the ankle. He was operating a small farm as well as working full time as a

FIG. 2-A

FIG. 2-B

Figs. 2-A through 2-D: Case 1. Follow-up roentgenograms made with the ankle in plantar flexion at fourteen weeks and twenty weeks gave a false impression of progressing bone union because the chip was viewed through the intervening intact dome. At nine months in the projection made with the ankle in dorsiflexion the fracture line was still visible. For correct interpretation of healing, the chip must be shown in profile on top of the dome.

Fig. 2-A: Three weeks after operation.

Fig. 2-B: Fourteen weeks after operation.

FIG. 2-C

Twenty weeks after operation.

FIG. 2-D

Nine months after operation.

laborer, carrying logs and brush on hillsides. Notwithstanding those trying conditions, the ankle performed normally. The fracture line was still visible on roentgenograms made March 1, 1954, five years after operation.

The good clinical result, together with the roentgenographic evidence of failure of bone union, was interpreted as showing that the patient had attained solid fibrous union which provided a useful, painless ankle. It was apparent that the eight weeks of immobilization had been inadequate for the formation of bone union.

CASE 3. J.R.R., a seventeen-year-old boy, had had no pain in his right ankle until November 19, 1954, when he injured it in a basketball game. The symptoms and signs were those of an inversion sprain. Roentgenograms showed a small chip fracture of the middle third of the lateral margin of the talar dome (Fig. 3-B). Immobilization in a walking cast for three weeks brought no improvement. A partially detached chip fracture of the dome was found at operation on December 13, 1954. The fragment, larger than the roentgenogram had indicated, was removed, and the resulting crater curetted. Immobilization in a cast was continued for five weeks. At the end of the sixth week the ankle was considered clinically normal. Nine months after operation the patient was free of symptoms.

CASE 6. E.J.S. was thirty-five years old and a tooler in an automobile-manufacturing plant. He had had no complaint of pain in his ankles until October 2, 1953, when he fell eighteen feet, from a ladder to the floor, injuring his back, pelvis, and ankles. He was taken to a hospital where he was found to have a fracture of the pelvis, a compression fracture of the second lumbar vertebra, and sprains of both ankles. With treatment at that hospital he recovered from the fractures of the spine and pelvis, except for occasional, residual aching in the lumbar region. He was not told of any ankle fracture and received no treatment for the sprains other than the bed rest necessitated by the fractures of the spine and pelvis. He returned to his work after discharge from the hospital.

One year after his accident he was seen at The Cleveland Clinic because of swelling and aching in the right ankle; both symptoms being relieved by rest, but aggravated by active motion. The left ankle remained free of pain unless used excessively to spare the right ankle. Examination showed the left ankle to be normal, but the right to be somewhat swollen, with

FIG. 3-A

FIG. 3-B

Figs. 3-A through 3-H: Roentgenograms showing the four stages of transchondral fractures of the talar dome—those of the lateral margin in 3-A, 3-C, 3-E, and 3-G in the left column and those of the medial margin in 3-B, 3-D, 3-F, and 3-H in the right column. In Figure 3-H the displaced chip is not seen clearly, but the lateral view of the same fracture (Fig. 15-B) shows it to occupy the anterior compartment of the ankle.

Fig. 3-A: Case 2. Lateral fracture, Stage One.

Fig. 3-B: Case 3. Lateral fracture, Stage Two.

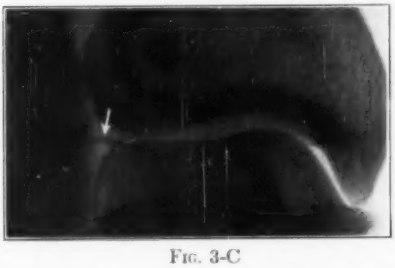

FIG. 3-C

Case 4. Lateral fracture, Stage Three.

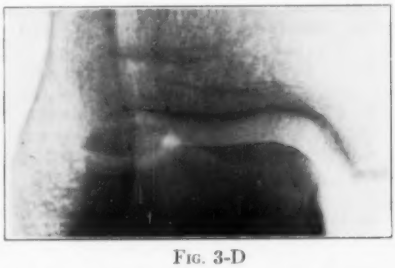

FIG. 3-D

Case 1. Lateral fracture, Stage Four.

FIG. 3-E

Case 11. Medial fracture, Stage One.

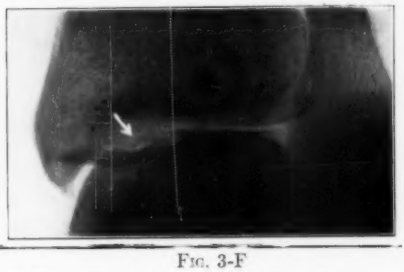

FIG. 3-F

Case 13. Medial fracture, Stage Two.

FIG. 3-G

Case 17. Medial fracture, Stage Three.

FIG. 3-H

Case 7. Medial fracture, Stage Four.

tenderness anterior to the lateral malleolus. The ankle dorsiflexed to 90 degrees and plantar flexed to 115 degrees. Roentgenograms made October 15, 1954, showed a normal left ankle.

There was an area of cortical elevation on the lateral margin of the right talus. In the roentgenographic report the possibility of osteochondritis dissecans was suggested and a small, loose body was noted in the medial margin of the ankle opposite the medial malleolus as well as an osteophytic spur that protruded from the anterior lip of the tibia (Figs. 14-C and 14-D).

Four months of conservative treatment with physical therapy gave no relief; operation was performed on February 22, 1955. A transverse osteotomy of the lateral malleolus gave wide exposure of the ankle joint. The osteocartilaginous fragment, measuring 2.5 by 1.3 by 1.3 centimeters, was detached from the crater in the middle third of the lateral margin of the talus. Two small, loose bodies were found—one lying between the talus and medial malleolus, and the other attached to the anterior inferior lip of the tibia. All three fragments were removed. The crater of the large fragment was curetted down to trabecular bone. The osteotomy was secured by a screw, and a toe-to-groin cast was applied. The pathological report read: "Necrosis of bone adjacent to cartilaginous cap. No evidence of healing at present".

Immobilization was continued for ten weeks. The patient returned to light work in August 1955, and to his regular work the following month. Six months after operation he had only occasional slight aching in the affected ankle and was not restricting its use.

Case 7. M.D.F., an eighteen-year-old laborer, had had no complaint concerning his left ankle until November 9, 1950, when, in jumping over a conveyor system, he caught his left heel and fell, striking the lateral aspect of the left ankle against a steel support. Roentgenograms showed a large defect in the middle third of the medial border of the talar dome and a large, loose body, eighteen by seven millimeters, occupying the anterior portion of the ankle joint (Figs. 3-H and 15-B). At operation, on November 28, 1950, the loose fragment was removed; it had healthy cartilage on one surface and raw trabecular bone on the other.

Seven months after operation the patient passed the physical examination for the United States Air Force, but for the next three years he continued to have some aching in the ankle on prolonged marching. Roentgenograms made two years after operation showed some filling in of the osseous defect of the talus and a small flake distal to the tip of the medial malleolus.

Case 11. M.C., a housewife, sixty-two years old, had had no disability in the right ankle until March 16, 1953, when she fell in a ditch, injuring the right foot and ankle. Roentgenograms showed a fracture of the calcaneus which was treated by immobilization and which healed without incident. No injury to the talus was noted. Fourteen months later, on May 3, 1954, the patient complained of pain in the right ankle. The pain was aggravated by walking but relieved by resting. Swelling and tenderness were present about the medial malleolus. The original roentgenograms were re-examined and were found to show a small lesion of the medial border of the talus (Fig. 3-E). Additional roentgenograms, including planigrams, showed the lesion still present, apparently increased in size. A diagnosis of osteochondritis dissecans was made. On June 2, 1954, arthrotomy of the right ankle showed a soft spot in the cartilage of the mid-portion of the medial border of the talar dome. The cartilage was intact. The soft spot was not incised or removed. A coincidental finding of stenosis of the sheaths of the peroneal tendons was treated by tenovaginotomy.

During the year following arthrotomy the patient continued to complain of the same symptoms that she had had prior to operation. Roentgenograms made on May 2, 1955, showed the lesion to be still present, and the roentgenographic diagnosis at this time was compression fracture.

The point of interest in this case was the healing of the calcaneal fracture although the talar fracture remained unchanged.

Case 13. P.P.M., a housewife, forty-seven years old, was treated for a ganglion of the peroneal tendon sheath of the right ankle. The ganglion was ruptured by digital pressure and did not recur. Routine roentgenograms made February 4, 1954, at the first visit, showed a fairly large area of osteochondritis dissecans of the medial margin of the talar dome (Fig. 3-F). The patient at first denied any injury to, or complaint of pain in, the ankle and declined treatment for the bone lesion. However, on October 13, 1955, when questioned again, she recalled that she had had instability of the ankle for years, that the ankle turned in while walking, and that she had occasionally experienced sharp, lightning pains in the ankle. She attributed these pains to arthritis and did not consider either the pains or the roentgenographic evidence to be serious enough to justify operation. Follow-up roentgenograms made September 30, 1955, showed the lesion unchanged in appearance.

Case 17. L.B., a beauty-parlor operator, thirty-four years old, complained of pain in the right ankle for six months, beginning in June 1951. She said that she had sprained the ankle severely in 1946 but had had no medical attention at that time. During the five-year interim the ankle was free of pain. In June 1951, with no further injury, she began to experience a sensation of "the bones in the right ankle seeming to slide over each other", and the foot inverted frequently, becoming progressively more painful until she could wear only low-heeled shoes. The foot was held in pronation, and the local skin showed numerous callosities. When the foot was forced in plantar flexion there was a suggestion of a palpable defect on

the medial margin of the superior surface of the talus. Roentgenograms made on January 2, 1952, showed a partly detached fragment on the medial aspect of the superior articulating surface of the talus, apparently under the posterior rim of the medial malleolus (Fig. 3-G). Operative removal of the chip was recommended, but the patient declined the suggestion.

The symptoms subsided during the following three months. Follow-up roentgenograms made on May 13, 1952, eleven months after the onset of symptoms, showed: "marked improvement in the appearance of the osteochondritic lesion with apparent loss of definition of the loose body and with resolution in the area of increased density."

On December 8, 1952, fourteen months after the onset of symptoms, the patient again complained of crepitus in the ankle. Roentgenograms made on December 15, 1952, were reported as showing no change from those of the previous May.

On March 2, 1954, thirty-three months after the onset of symptoms and twenty-seven months after the first roentgenograms, re-examination showed the lesion to have the same appearance as in the first examination. The state of healing was not found to be as reported at the interim examinations.

Until May 1954, the patient continued to have intermittent and variable discomfort in the ankle, but was able to continue working.

In this patient the time from original injury to follow-up was eight years; symptoms were present for thirty-five months; and there was roentgenographic proof of the existence of the lesion for twenty-seven months.

CASE 18. On September 12, 1949, H.R., a tabulator-machine operator, thirty-three years old, complained of pain of two months' duration in the left ankle. She denied injury and said that the pain had been gradual in onset and aggravated by walking. Adhesive strapping had given partial relief. Both feet were pronated; there was tenderness over the anterior aspect of the left ankle and slight swelling of the lower third of the leg and the ankle. Range of motion was from 90 to 130 degrees. Roentgenograms were reported as showing osteochondritis dissecans of the medial border of the left talar dome. Conservative treatment by leg cast for six weeks brought no improvement.

Two months after the first visit the patient began to complain of occasional "catching" pain in both ankles. Roentgenograms made on November 18, 1949, showed osteochondritis dissecans of the superior surfaces of both tali (Fig. 15-C). Bilateral intermittent pain continued. By March 6, 1950, the pain in the left ankle had become intolerable. Arthrotomy, March 8, 1950, showed a loose body, one centimeter in diameter, in the posterior portion of the left ankle joint. Cartilage over the posterior portion of the medial border of the talar dome was eroded. The cartilage over a small area anterior to this was soft and boggy; this was removed and the underlying bone lightly curetted. The remainder of the joint looked normal. A boot cast was employed for seven and one-half weeks. Subsequent range of motion gradually increased until full movement was present eleven months after operation. During the next two years there were no symptoms referable to the left ankle.

The right ankle, however, continued to be painful at intervals, with increasingly frequent episodes of "catching", stiffness, and aching. By December 1951, locking occurred repeatedly. Arthrotomy of the right ankle, March 5, 1952, showed a loose body, covered by fibrillated soft cartilage, on the medial border of the talar dome. The fragment, which measured one by two centimeters, was removed. Recovery was slower than with the left ankle. Intermittent discomfort and swelling occurred during the next two years (Fig. 15-D). The surgeon in charge of this patient suggested that the persistence of symptoms might be due to one or more small bone fragments still present in the joint cavity.

In an interview with the patient on March 6, 1954, she was asked if she could recall any injury to the ankle, previous to 1949. She then recalled that she had had painful ankles throughout her life; that she had always had to wear low-heeled shoes; that one foot had been in inversion during one winter, when she was a child; and that in 1931 she had fallen off the suspended rings in her high-school gymnasium, spraining her right ankle so severely that she limped for four months. From 1944 until 1947 her work required standing, which caused continuous pain in both ankles—worse in the afternoon and better after a night's rest. For the two years immediately preceding the date of onset, as given by her during the first examination, she had had a sedentary occupation and less discomfort in the ankle. During her original examination, when asked about the occurrence of trauma, she gave a negative answer because she thought the question referred only to recent trauma.

CASE 23. W.C., a man, forty-seven years of age, had no complaint concerning his left ankle until August 15, 1957, when, descending from a truck, he set his foot on a small rock which turned with him and sprained his ankle.

When examined at the hospital immediately after his injury there was tenderness over the posterior fasciculus of the deltoid ligament and over the entire lateral malleolus, together with a moderate degree of swelling of the ankle and limitation of motion of the foot in both plantar and dorsal flexion. There was no other associated injury.

Roentgenographic examination made at the time of the first visit showed a tiny chip of

TABLE I

ANALYSIS OF TWENTY-FOUR CASES OF LESIONS OF THE DOME OF THE TALUS

Case number	1	2	3†	4	5	6†	7†	8†	9	10	11
Sex of patient	M	F	M	M	M	M	M	F	NR*	M	F
Age at onset of symptoms (*years*)	30	62	17	32	36	35	18	52	NR	30	62
Duration of symptoms (*months*)	0	0	0	0	0	14	0	36	NR	96	14
History of trauma	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	NR	Yes	Yes
Type of symptoms (*sprain or arthritis*)	Spr.	Spr.	Spr.	Spr.	Spr.	Arthr.	Spr.	Arthr.	NR	Arthr.	Arthr.
Ankle affected (*left or right*)	L	R	R	R	R	R	L	R	L	R	R
Site of lesion (*medial or lateral*)	Lat.	Lat.	Lat.	Lat.	Lat.	Lat.	Med.	Med.	Med.	Med.	Med.
Stage of lesion ** (*1, 2, 3, or 4*)	4	1	2	2	3	3	4	2	1	2	1
Diagnoses recorded (*fracture, osteochondritis dissecans, or both*)	Both	Fract.	Fract.	Fract.	Fract.	Both	Fract.	Both	Both	Both	Both
Concomitant injuries	No	No	No	No	No	Yes	No	No	NR		Yes
Conservative treatment:											
Duration of treatment (*months*)	0	½	¾	2	2	16	⅔	36	NR	96	14
Clinical results of treatment	0	?	Poor	Good	Good	Poor	Poor	Poor	NR	Fair	Poor
Operative treatment:											
Type of operation (*arthrotomy only, removal of chip, or replacement of chip*)	Replace.	0	Remov.	0	0	Remov.	Remov.	Remov.	0	0	Arthr.
Pathological examination	0	0	Yes	0	0	Yes	Yes	NR	0	0	0
Subsequent immobilization (*months*)	2	0	1¼	0	0	2½	NR	1½	0	0	NR
Clinical results of treatment	Good	0	Good	0	0	Good	Good	Good	NR	Fair	Poor
Follow-up duration (*months*)	60	½	9	2	2	6	58	7	NR	0	12
X-ray appraisal of results	Good	0	Good	Good	Good	Good	Good	NR	Fair	0	Poor

* Items that were not recorded in the original histories are indicated by NR.

** One feature not included in tabulations in previous reports is the stage of lesion. This is an arbitrary classification which was developed to aid understanding of the etiological mechanism of the fracture and to help in determining the appropriate treatment. The stages are:

Stage One—a small compression fracture.

Stage Two—incomplete avulsion of a fragment.

Stage Three—complete avulsion without displacement.

the bone below the medial margin of the articulating surface of the talus, apparently coming from the medial margin of the talus. Treatment was by compression dressing for three and one-half weeks. There was no improvement during this time, and the patient was admitted to the hospital on September 10, 1957.

Arthrotomy of the ankle was performed on September 11 through a posterior approach, without osteotomy of the malleolus. A small depressed chip fracture of the posteromedial aspect of the dome of the talus was found. This was removed by a bone gouge and the articular cartilage about the edge of the defect, approximately one-half of an inch in diameter, was beveled. A compression dressing without cast immobilization was applied.

Postoperative progress was slow because of persistent edema of the lower leg and foot. The patient returned to work as a highway laborer on March 24, 1958.

The pathological report, which included the scrapings from the crater, showed cartilage and cancellous bone fragment exhibiting some fibrosis and osteoblastic activity. Roentgenographic appraisal showed satisfactory postoperative appearance, and the clinical course was deemed good.

CASE 24. G.H., a boy, eighteen years old, was first seen on November 27, 1956. At age sixteen, while playing high-school basketball, he sprained his right ankle badly; he had an identical injury one year later. The patient had pain, swelling, and crepitus in the ankle after activity; these symptoms subsided with rest. The only treatment was elastic bandage and application of heat. There were no concomitant injuries.

Examination showed a complete range of motion with loud crepitus on motion; there was no swelling or tenderness at the time of examination.

TABLE I — *Continued*

12	13	14†	15	16	17	18 F†		19†	20†	21†	22†	23	24
M	F	F	M	F	F			M	M	M	M	M	M
NR	47	27	15	70	34	33	33	14	24	10	18	47	21
3	"Years"	½	12	¾	6	"8"	"8"	1	120	12	24	0	0
Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes
NR	Arthr.	Spr.	Arthr.	Spr.	Arthr.	Arthr.	Arthr.	Spr.	Arthr.	Arthr.	Arthr.	Spr.	Spr.
R	R	L	R	R	R	L	R	R	L	L	R	L	R
Med.	Med.	Med.	Med.	Med.	Med.	Med	Med.	Lat.	Med.	Med.	Lat.	Med.	Lat.
1	2	1	2	2	2	4	3	2	3	4	4	2	2
OD	OD	OD	OD	Both	OD	OD	OD	OD	OD	OD	Both	Fract.	Fract.
	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes			No	No
3	0	1	18	¾	33	9	19	5	122	14	24	1	0
NR	Fair	Poor	Fair	Poor	Poor	Poor	Poor	Poor	Poor	Poor	Poor	Poor	0
NR	0	Remov.	0	0	0	Remov.	Remov.	Remov.	Remov.	Remov. twice	Remov.	Remov.	Remov.
NR	0	NR	0	0	0	NR	NR	NR	NR	NR	Yes	Yes	0
NR	0	NR	0	0	0	2	NR	¾	2¼	¼	2¼	0	1
NR	0	Good	0	0	0	Good	Good	Good	Good	1–Poor 2–Good	Good	Good	Good
NR	20	16	18	0	33	48	24	50	3	48	22	11	4
NR	0	Good	Poor	NR	Poor	Good	Good	Good	Good	Good	Good	Good	Good

Stage Four—avulsed fragment displaced within joint.

† We do not endorse the use of the criteria good, fair, or poor with reference to the appearance of the empty crater in postoperative roentgenographic evaluation of patients in whom the fragment was removed, since the fibrous filling of the crater cannot be seen in films. The chief value of the postoperative roentgenogram is the proof of presence or absence of small bone fragments overlooked at operation. The evaluations of such cases in Table I were those reported by the roentgenologists, and have been included only to permit comparison with Table II of Rödén, Tillegård, and Unander-Scharin.

Roentgenographic examination showed an avulsion fracture of the anterior aspect of the lateral border of the talus, a Stage Four fracture.

Operation, performed on December 4, 1956, consisted in removal of the multiple fragments and osteotomy of the fibula to give access to the ankle joint. A toe-to-groin cast was applied.

The patient returned to work, operating a shoe-making machine, on January 25, 1957. The cast was removed on February 8, 1957. Four months after operation he had no complaints concerning the ankle. All symptoms had been relieved and he was participating in amateur athletics without disability.

The characteristic features of the twenty-four patients with twenty-five osteochondral fractures of the superior surface of the dome of the talus are listed in Table I.

REVIEW OF THE LITERATURE

Our review of medical literature on the subject of osteochondral lesions of the talus has produced reports of two cases of loose bodies in the ankle joint, thirty cases of flake fractures, and 151 cases of osteochondritis dissecans—183 cases in all. Eight of the patients with osteochondritis dissecans had bilateral ankle lesions, giving a total of 191 fractures. These reports are listed in Table II.

TABLE II

PREVIOUSLY PUBLISHED CASES OF TRANSCHONDRAL FRACTURES OF THE TALAR DOME

Entity	Authors	Year	No. of Cases
Loose Bodies	Monro	1856	1
	Stevenson	1925	1
	Total		2
Osteochondritis Dissecans	Kappis	1922	1
	Wolff	1926	2
	Breitländer	1927	1
	Cordes	1927	1
	Harms	1927	1
	Vogel	1927	1
	Deutschländer	1928	1
	Axhausen	1928	1*
	Diaz Garcia	1928	1
	Läwen	1929	4
	Sidler	1929	1
	Radicke	1930	1
	von Staa	1930	1
	Fairbank	1933	1
	Pérez Duefio	1933	2
	Hellström	1934	1
	Ribbing	1937	1
	Myhre	1939	3
	Hoffmann	1939	1
	Burr	1939	1
	Brickey and Grow	1940	1
	Mensor and Melody	1941	1
	Eskesen	1942	1
	Lewis	1942	1
	Aronsson	1942	4
	Cobey	1943	3
	Cohen	1943	1
	Dawson	1943	1
	Hutchison	1943	2
	Lagomarsino	1944	1
	Wilner	1946	1
	Vaughan and Stapleton	1947	4
	Janes	1947	1*
	Lavner	1947	1
	Ray and Coughlin	1947	13*
	Eberhard	1947	3
	Soeur	1950	1
	Simpson	1950	1
	Howorth	1952	1
	Green and Banks	1953	1
	Rödén, Tillegård, and Unander-Scharin	1954	55**
	Anderson and LeCocq	1954	6
	DeGinder	1955	19
	Cameron	1956	1
	Total		151
Flake Fractures	Rendu	1932	1
	Eskesen	1942	6
	Wilner	1946	1
	Kleiger	1948	8
	Marks	1952	1
	Coltart	1952	1
	Nisbet	1954	3
	Bosien, Staples, and Russell	1955	9
	Total		30
	Grand Total, Cases		183
	Grand Total, Fractures		191

* 1 bilateral case each
** 5 bilateral cases

NOMENCLATURE

The lesions discussed herein have been classified and reported under many different names. None of the names presently in use is at the same time accurate, comprehensive, and specific. Since the fracture concerned is a specific type with individual characteristics that set it apart from other fractures, it deserves both recognition and a distinctive name. The name proposed for this entity is *transchondral fracture*, a name which is correct from both anatomical and etiological points of view.

TRANSCHONDRAL FRACTURES

Definition

A transchondral fracture is a fracture of the articular surface of a bone, produced by a force transmitted from the articular surface of a contiguous bone across the joint and through the articular cartilage to the subchondral trabeculae of the fractured bone. Either of two physical types of fracture may result. First, the fracture may result in a small area of compressed trabeculae, with or without demonstrable damage to the overlying cartilage. Or it may be an avulsion of an osteocartilaginous flake.

The transchondral fracture differs from other small compression or chip fractures because of several unique characteristics. It is difficult to recognize clinically

as a fracture since it may occur as a result of trauma so trivial that it passes unnoticed, or its presence may be masked by more painful nearby soft-tissue injuries. The compressed or avulsed segment of bone has no soft-tissue attachments, no blood supply, and no nerve supply, and is consequently unusually susceptible to the development of avascular, aseptic necrosis.

Incidence

Numerical incidence: Two hundred and fourteen transchondral fractures of the dome of the talus have been identified. This figure is certainly too small. It has been necessary, on the grounds of accuracy, to exclude cases of chip or avulsion fractures of the talus that have not been described or located with sufficient pre-

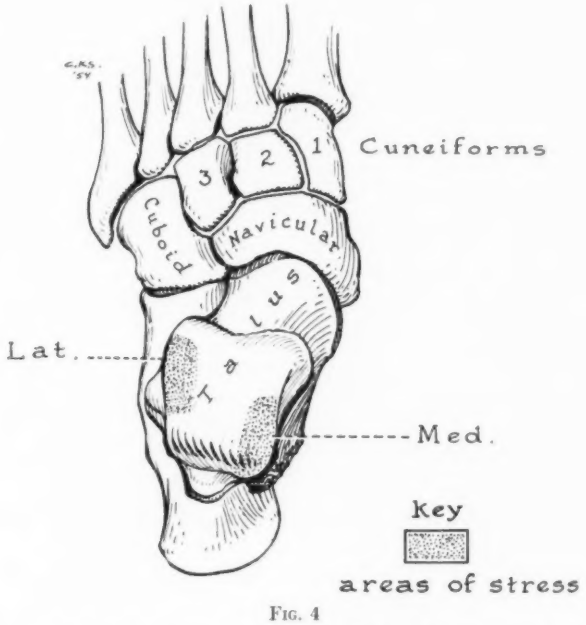

FIG. 4

Drawing showing the usual sites of transchondral fractures of the talar dome. Of 201 cases there were eighty-eight fractures in the lateral border (43.7 per cent), most of which were located in the middle third of the border. The medial border was involved in 113 fractures (56.3 per cent), usually in the posterior third of the border.

cision to make certain their identity. Coltart [19] reported fifty-six chip or avulsion fractures of the talus, but located only one. Lipscomb and Ghormley [49] reported sixteen but located none. Wilson [81] reported four cases but located none. Boyd and Knight [8] gave no statistics. Only a short abstract of Brewer's report [11] could be obtained *.

Comparative incidence: Coltart [19], reviewing statistics of the Royal Air Force from 1940 to 1945, listed a total of 25,000 fractures of all bones. Of this number, 4,000 affected the ankle and foot. There were 114 cases of fracture of the

*Three cases that were included in previous reviews of flake fractures of the ankle are not included in the present study on the ground that they did not concern fractures of the dome of the talus: Schnaberth's [65] lesion was in the subtalar joint, Schosserer's [66] fracture was of the anterior corner of the tibia, and Schulte-Tenckhoff's [67], from the lateral malleolus. The two lesions reported by Schulte-Tenckhoff draw attention to the fact that the margins of the talus usually give way before the impacting areas of the tibia or fibula are damaged.

talus of which fifty-six were chip or avulsion fractures. Wilson listed seventeen fractures of the talus, of which four were chip or sprain fractures but, since he specified that these were caused by ligamentous avulsion, it is doubtful if his cases should be included. Lipscomb and Ghormley [49] reported 114 fractures of the talus, of which sixteen were chip fractures. On the basis of these statistics, approximately 0.09 per cent of all fractures are transchondral fractures of the talus.

Bosien, Staples, and Russell [7], in 1955, reviewed a series of ankle sprains in men in two college classes. They reported that 113 patients had incurred 133 sprains. In this number they identified nine cortical avulsion fractures of the talus, an incidence of 6.75 per cent.

Anderson and LeCocq [1], in 1954, reported osteochondritis dissecans in six of twenty-seven cases of sprains of the lateral collateral ligament of the ankle.

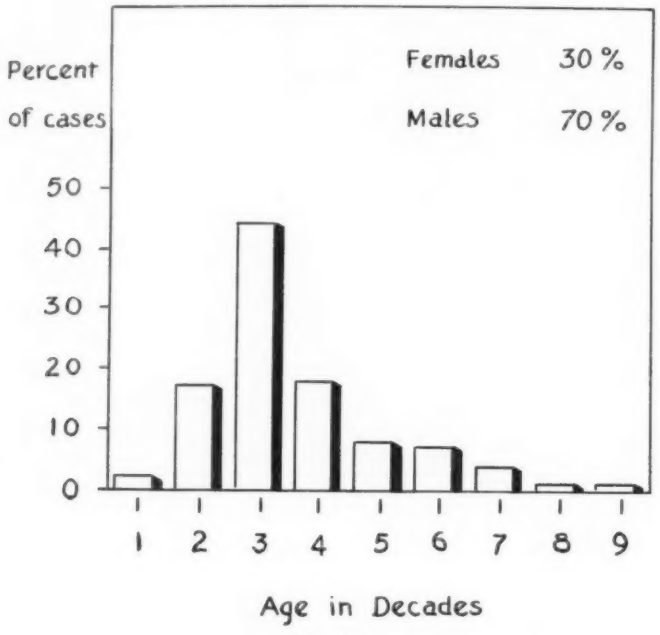

CHART 1

The incidence of transchondral fractures on the basis of sex and age. The sex of the patient was mentioned in 173 cases, of which there were 123 male and fifty female patients. The age of the patient was recorded in 164 cases; the peak incidence was in the third decade.

Sex incidence: Of the 214 fractures reviewed, the sex of the patients was given in 174 cases: male, 124 cases (approximately 70 per cent); female, fifty cases (30 per cent). Most of the cases included in these figures were reported from military sources which could be expected to have a predominantly male population.

Age incidence: The patient's age was reported in 165 cases. The youngest patient was five years old, the oldest was eighty. The age incidence, by decades, is shown in Chart I.

Ankle affected: Of 166 fractures, the right talus was affected in eighty-nine patients (53.6 per cent), the left talus in seventy-seven (46.4 per cent).

Site of lesion: Of 201 fractures in which the site was described in the text or shown in roentgenograms, the lesion involved the lateral border of the talus

in eighty-eight patients (43.7 per cent) and the medial border in 113 (56.3 per cent) (Fig. 4).

Stage of lesion: The stage of the lesion was indicated in 100 patients: Stage One, 15 per cent; Stage Two, 36 per cent; Stage Three, 28 per cent; and Stage Four, 21 per cent.

Etiology

The etiological mechanism that produces a fracture is usually obvious. The symptoms and roentgenographic appearance are characteristic. Treatment by reduction and adequate immobilization ordinarily results in healing, but if immobilization is incomplete or of too short a duration, delayed union or non-union ensues and may last indefinitely.

The mechanism that causes osteochondritis dissecans, on the other hand, has been a matter of dispute for almost seventy years. In 1931 Wagoner and Cohn [77] made a complete review of the literature up to that time. They listed sixty-four reports; trauma was noted as the causative agent in forty-one. The remaining twenty-three offered eighteen causes other than trauma; of these eighteen causes only one, arthritis, was acceptable enough to be selected by four authors.

Most of the theories of causation, other than that of trauma, have been discarded on the grounds of lack of evidence, but some continue to have their advocates. The hereditary factor, favored by Wagoner and Cohn, was revived in 1955 by Gardiner [31] and Pick [57]. However, only six families were reported as having osteochondritis dissecans in more than one of their members, a fact which makes the hereditary theory statistically untenable.

The theory of spontaneous necrosis has had strong supporters whose chief point of argument has been the fact that no other etiological factor has been completely proved.

The theory of trauma as the etiological factor has had the greatest number of adherents and has become increasingly popular. Yet some of the authors who espouse the case for trauma say that the exact mechanism is still unknown and they have been hesitant to identify osteochondritis dissecans as a fracture. Rödén and his associates [63] reported fifty-five cases and attributed all of their lateral-margin lesions, except one, to trauma, noting that 61 per cent of their forty-one patients with medial-margin lesions had had trauma. DeGinder [24] attributed all of his nineteen cases to trauma.

Lesions with identical characteristics have been called fractures by some authors and osteochondritis dissecans by others. Eskesen [28] and Wilner [80] presented cases of fractures and of osteochondritis dissecans as different entities, but failed to indicate the distinctive features.

Why have not all the cases been called fractures? The answer lies in the fact that it has not been demonstrated clearly that the diagnosis of fracture could be compatible with three characteristics that usually do not pertain to fractures: (1) that a fracture could occur without recognizable trauma, (2) that the resulting fracture could be painless, and (3) that proper reduction followed by immobilization for a reasonable length of time would not produce bone union. Each of these three characteristics appears to be at odds with one of the basic concepts of fractures.

If fractures embodying all three of these characteristics could be produced experimentally or confirmed by clinical evidence, there should remain no reasonable objection to classifying the lesions of osteochondritis dissecans as osteochondral fractures.

Anatomical Investigation

Purpose: Experiments were undertaken with the hope of discovering whether typical lesions of osteochondral fractures and osteochondritis dissecans of the

dome of the talus could be produced in the laboratory by applying various forces to the ankles of amputation specimens.

Materials: Preliminary experiments using the limbs of cadavera from the dissecting room were unsatisfactory, owing to the age and nutritional condition of the specimens and the alterations in the relative strength of the bone and soft parts caused by the materials used for preservation. Accordingly, above-the-knee specimens were used—ten after temporary preservation by refrigeration, and five immediately after operation. The freshly amputated specimens proved to be the most satisfactory.

Methods: Some difficulty was experienced in holding the foot in the proper position while force was applied to the knee and thigh. The most satisfactory method involved the use of a heavy block of wood made with a deeply slanting groove in the center (Fig. 5). The foot of the specimen was placed in the groove

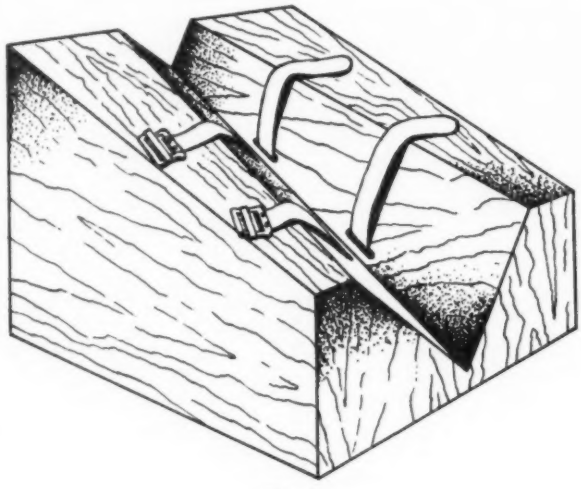

FIG. 5

Drawing of the wooden block used in anatomical experiments to secure the foot of the amputation specimen during manipulation of the knee and thigh. The inclination of the groove made it possible to immobilize the foot with the ankle in any desired position.

and strapped tightly in the desired position. The slant of the groove and the inclination of the sides permitted a choice of any combination of inversion or eversion with plantar flexion or dorsiflexion of the foot, with respect to the vertical lower leg.

The block, holding the foot, was placed on the floor and held immobile by one of us. The other stood beside the block, rested his left hand upon the knee of the specimen which was flexed to 90 degrees, leaned forward with chest upon the left hand to put his body weight on the knee, then with the right hand maneuvered the thigh of the specimen into further inversion, eversion, or rotation of the ankle.

When, during the application of force, the normal range of motion of the joint was exceeded, a cracking, crunching sound was emitted by the ankle, caused by tearing of the collateral ligaments and crushing of the trabeculae of the dome. The ankle then was dissected free and examined for damage. In some experiments no damage to the talus could be found, although the collateral ligaments were torn. In examining the talus it was necessary to recall that apparently undamaged articular cartilage could conceal a Stage One fracture. A report of no damage to the talus should not be made until the talus has been sectioned through the suspected area.

TABLE III

RESULTS OF ANATOMICAL EXPERIMENTS

Type of Manipulation	No. of Specimens	Experimental Fractures Produced	
		Lateral Border	Medial Border
Recent (Refrigerated) Amputation Specimens			
Eversion + dorsiflexion	1		
Eversion + plantar flexion	1		
Inversion + dorsiflexion	2	1	
Inversion + plantar flexion	2		
Inversion + plantar flexion + rotation	4		1
Fresh Amputation Specimens			
Inversion + plantar flexion + rotation	5		1
Totals	15	1	2

The high rate of failure of the methods which occasionally produced fractures was due to the fact that only one experiment could be performed upon one specimen. Dissection of the ankle to determine the condition of the talus rendered the specimen unfit for further testing. Perhaps roentgenographic examination of the specimen after the application of force would have enabled us to determine the absence of fracture and have permitted us to remanipulate specimens in which the first attempts failed to produce fractures.

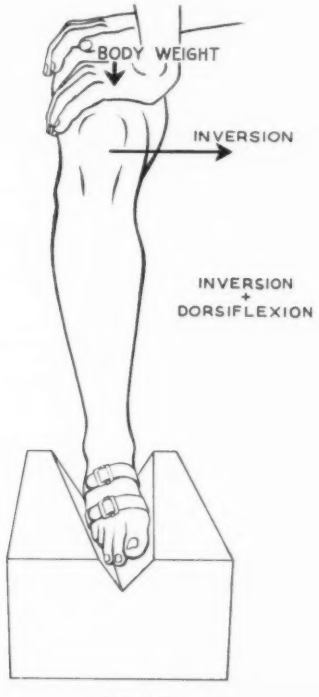

FIG. 6-A

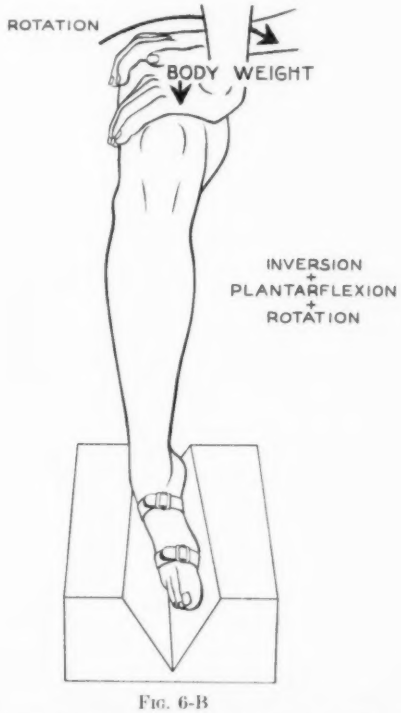

FIG. 6-B

Figs. 6-A and 6-B: Drawings illustrating the methods of manipulation of amputation specimens.

Fig. 6-A: The manipulation that produced fracture of the lateral border of the talus.

Fig. 6-B: The method of producing fracture of the medial border.

Lateral Lesion

A review of the operative findings in clinical cases of lesions of the lateral talar border showed that the following conditions usually were attendant upon the lesion: the lesion was found in the middle or anterior half of the lateral border, and damage was limited to two sites—the talar lesion and the lateral collateral ligament. There was no damage to the malleoli, to the medial collateral ligament, or to the interosseous (tibiofibular) ligament. Any lesion produced by experimental methods must produce the same, and no other, injury.

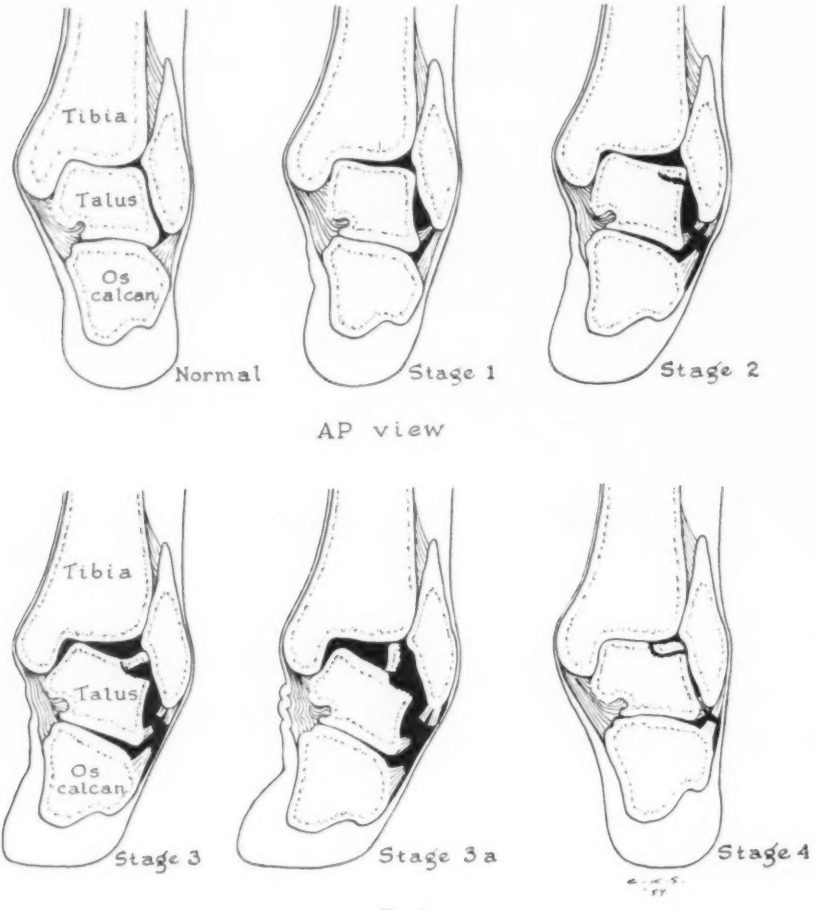

FIG. 7

Diagram showing the etiological mechanism of transchondral fractures of the lateral border of the talar dome. As the foot is inverted on the leg the lateral border of the dome is compressed against the face of the fibula (Stage One), while the collateral ligament remains intact. Further inversion ruptures the lateral ligament and begins avulsion of the chip (Stage Two), which may be completely detached but remain in place (Stage Three) or be displaced by inversion (Stage Four).

In these experiments the damage just described could be inflicted only by the maneuver of strong inversion of the dorsiflexed ankle (Figs. 6-A and 6-B).

In dorsiflexion of the ankle the wider anterior half of the talar dome was moved backward into the mortise, filling it snugly. In this position lateral rota-

tion in the frontal plane of the talus was impossible when only a moderate inverting force was applied. With the use of excessive inverting force, the talus within the mortise was rotated laterally in the frontal plane, impacting and compressing the lateral talar margin against the articular surface of the fibula, producing a small area of indentation in the talar margin—a Stage One lesion. If rotation were continued, a portion of the talar margin remained impacted against the fibula and was sheared off from the main body of the talus. This mechanism is illustrated diagrammatically (Fig. 7) and confirmed clinically (Figs. 3-A through 3-D).

This mechanism produced the experimental fracture of the anterior third of the lateral talar border as shown in Figure 8—a Stage Two lesion.

The importance of the lateral collateral ligament of the ankle in this mechanism should be noted, for it explains the difference in symptoms between the first

FIG. 8

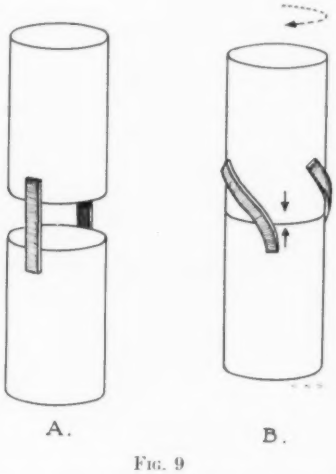

FIG. 9

Fig. 8: Photograph of transchondral fracture of the lateral border of the talar dome which was produced experimentally in an amputation specimen by forcible inversion of the dorsiflexed ankle. The fractured fragment, incompletely avulsed, is a Stage Two fracture of the anterolateral margin.

Fig. 9: Diagram illustrating the principle of torsional impaction as applied to a diarthrodial joint. In *A* the bones are represented by cylinders, the joint space is exaggerated for clarity, and the collateral ligaments are shown as thongs. In *B*, rotation of the upper cylinder on the fixed lower cylinder changes the direction of the ligaments, shortening their effective lengths and drawing the cylinders together until impaction occurs. In the ankle joint where the ligaments are taut at the beginning of rotation, only a few degrees of rotation are necessary to produce great impacting force.

and second stages of the lateral fracture. In the first stage the compression of the talar margin took place before the lateral ligament was stretched to the point of rupture. As a result, a clinical Stage One lesion would be almost painless, the symptoms of sprain being absent or minimal, and the compression fracture being painless in itself owing to the absence of sensory nerve endings in the articular cartilage and the minimal sensation in the cancellous bone which the cartilage overlaps [5,9,15,20,21,32].

Before continuation of the rotation of the talus could produce the second stage of the lesion, the middle and anterior fasciculi of the lateral collateral ligament had to rupture, as was found in the patient in Case 1 of this series. Fractures of the second, third, and fourth stages, all painless in themselves, then must be accompanied in clinical cases by the symptoms of acute inversion sprain: pain over the torn lateral ligaments.

In these experiments no injury to the malleoli, to the deltoid, or to the interos-

seous ligaments was found. Attempts to produce the lateral lesion by any other combination of movements of the ankle were fruitless. From our preliminary studies of the ankle joint in motion, utilizing both intact and coronally sectioned specimens, it appeared that eversion could play no part in the production of either lateral or medial fractures because of the shortness and obliquity of the articular face of the medial malleolus, and the strength of the middle fasciculus of the deltoid ligament. We, therefore, did not sacrifice specimens needlessly in eversion manipulations.

Medial Lesions

In a review of the clinical features of medial-margin lesions the following points were noted. In some cases no history of trauma could be identified (in thirty-three of 183 cases—18 per cent—no trauma was reported). Most authors reported that the patient's injury had been caused by inversion or adduction of the foot, but four cases (Harms'[34] case, Burr's[13] first case, Ray and Coughlin's[59] ninth case, Case 14 of the present series) occurred when the patient attempted to extricate the foot that had been caught by an external weight lying across the instep. Roentgenograms showed that the medial lesions, with one exception, were in the posterior half of the medial margin, that they appeared to be narrow in their anteroposterior diameter, that they were under the posterior inferior lip of the tibia, and that in some cases that lip of tibia appeared to have been indented upward (Fig. 15-A).

Fig. 10: Photograph of transchondral fracture of the medial border of the talar dome which was produced experimentally in an amputation specimen by employing, in sequence, the forces of plantar flexion, inversion, and external rotation of the tibia on the talus. The compressed area, a Stage One fracture, is situated in the posterior third of the medial border, the site where most of the medial fractures have been found in clinical cases.

FIG. 10

These points suggested that the mechanism might be: (1) plantar flexion of the ankle, (2) slight anterior displacement of the tibia upon the talus, (3) inversion of the ankle, and (4) lateral rotation of the tibia upon the talus (Fig. 6-B). With the employment of this sequence of forces, the following events were noted to take place in the ankle joint.

In plantar flexion of the ankle the narrow posterior half of the talar dome occupied the mortise somewhat loosely, owing to relaxation of the collateral ligaments, and lateral rocking of the talus in the mortise was permitted. Inversion of the talus turned its medial border uppermost so that the articular surface of the tibia rested only upon the ridge formed by the medial border of the talus. Slight forward displacement of the tibia on the inverted talus then removed from contact with the talar border all of the tibial articular surface except the posterior inferior lip. At this stage the two bones touched each other only at the intersection of two narrow ridges, the total area of contact amounting to approximately one-sixteenth of a square inch, or one-sixteenth of the normal area of articular contact. This raised the pressure in this small area to sixteen times the normal, jeop-

ardizing the integrity of this small, thrust-bearing portion of the talar surface.

The impacting force was further increased by the external rotation of the tibia on the talus. This caused the posterior lip of the tibia to move medially, grinding the posterior tibial lip across the talar border until the collateral ligaments became taut. When all slack of the collateral ligaments was fully taken up, further rotation of the tibia introduced an additional and powerful force of impaction owing to

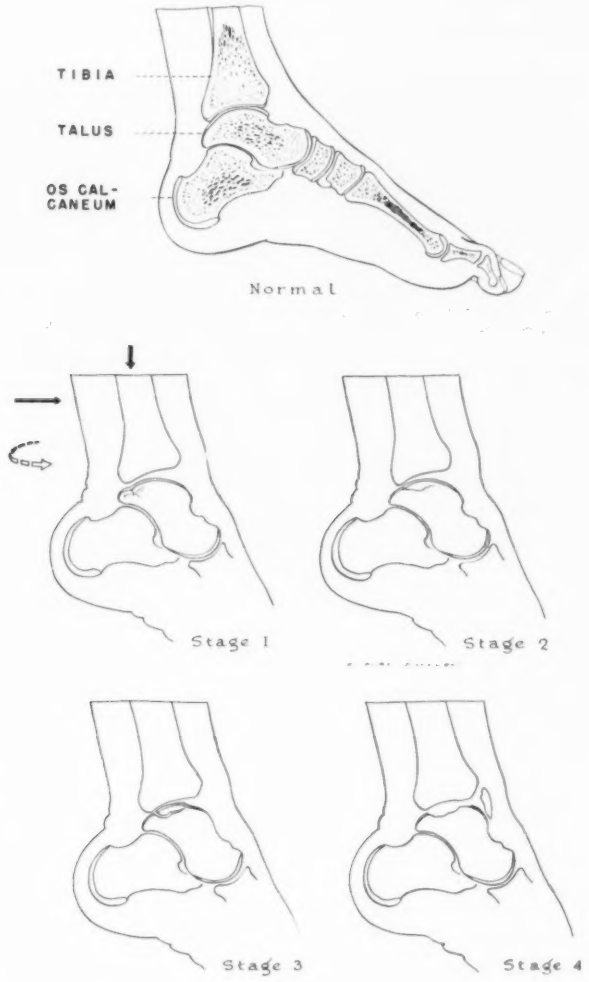

FIG. 11

Diagram showing etiological mechanism of transchondral fractures of the medial border of the talar dome. Plantar flexion of the foot with inversion, followed by rotation of the tibia on the talus produces a small area of compression (Stage One), the collateral ligaments remaining intact. Greater force causes the posteroinferior lip of the tibia to ride medially across the upturned medial margin of the talus, gouging out an osteochondral chip which is partially detached (Stage Two), completely detached (Stage Three), or displaced within the joint (Stage Four). The second stage usually is accompanied by rupture of the posterior fasciculus of the deltoid ligament or of the anterior fasciculus of the lateral collateral ligament.

the change in direction of the fibers of the collateral ligaments from vertical to spiral, shortening their effective length and drawing the tibia and talus strongly together.

This mechanism might be called rotational or torsional impaction. According to physicists and engineers it has no other name. It resembles, somewhat, the action of a screw being used to join two pieces of wood: when the pieces come into contact, further rotation of the screw through only a few degrees produces a tremendous force of impaction. (An effective nutcracker can be made to illustrate this mechanism by joining two wooden cylinders together end-to-end by leather

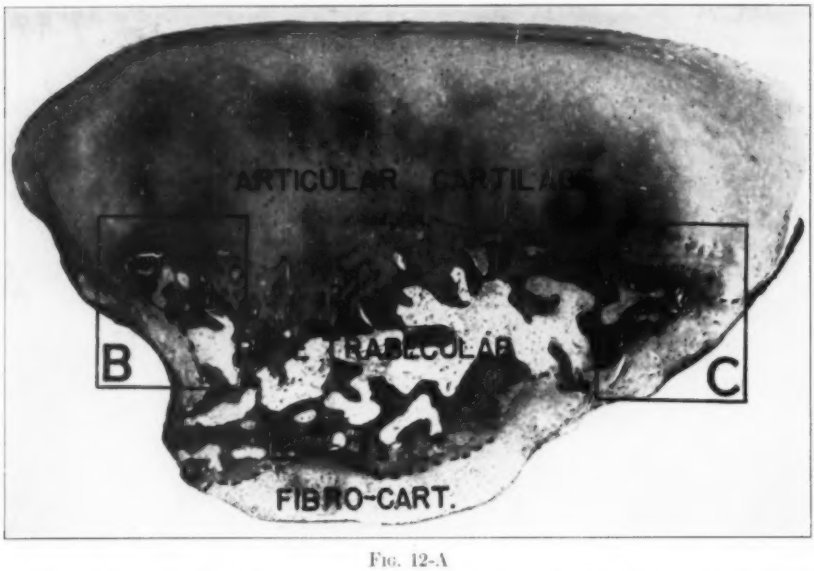

FIG. 12-A

Case 22. Photomicrograph (× 9) of cross section of a transchondral-fracture chip from the talus. This free fragment was removed from the anterolateral compartment of the right ankle of a boy, eighteen years old, who had symptoms of two years' duration (Table I). The fragment had originated from a crater on the lateral border of the dome of the talus which, at operation, was filled with firm fibrous tissue.

The large fragment had an articular surface of hyaline cartilage. The entire fracture surface (broken line) was overgrown with firm fibrous and fibrocartilaginous tissue.

thongs parallel to the long axes of the cylinder. By rotating the cylinders in opposite direction, the adjacent ends are impacted forcefully.) Torsional impaction occurs in any diarthrodial joint when one bone is fixed and the other rotated upon its long axis (Fig. 9).

With the knee flexed to 90 degrees the mechanical advantage of the body weight is forty to one, the short arm being the radius of the tibia (one centimeter), the long arm being the length of the femur (forty to fifty centimeters). This mechanical system makes it possible for damage to occur to the medial margin of the talus even though the rotation is stopped short of the point of tearing the posterior fibers of the deltoid ligament. The force of torsional impaction appears to be the principal agent in the production of the medial-margin Stage One lesion, the so-called atraumatic osteochondritis dissecans, when applied to the previously plantar-flexed, inverted ankle. Clinically observed cases of this lesion have been characterized by a lack of history of trauma, owing to the painlessness of the bone lesion and the fact that the collateral ligaments had not been torn.

Further rotation of the tibia on the talus beyond the production of the Stage One lesion presumably resulted in rupture of the posterior fibers of the deltoid

ligament. The resultant freedom permitted more forward motion of the lip of the tibia upon the edge of the talus. This movement tended to shear off or gouge out a fragment of the talus to produce a Stage Two or Stage Three lesion, or if the fragment was displaced into the anterior compartment of the joint, a Stage Four lesion. This mechanism is shown in diagrammatic form (Fig. 11) and in roentgenograms of clinical cases (Figs. 3-E, 3-F, 3-G, 3-H, and 15-B).

In the clinical cases, Stages Two, Three, and Four have been marked by tenderness and pain over the posterior fasciculus of the deltoid ligament.

This mechanism explains satisfactorily the findings in the clinical cases. The

FIG. 12-B

FIG. 12-C

Figs. 12-B and 12-C: Different areas clearly indicate living cells in hyaline cartilage, fibrous tissue, and fibrocartilaginous tissue. The lacunae in the bone trabeculae are vacant and the osteocytes are dead.

In such intra-articular loose bodies—without blood supply and nourished only by synovial fluid—hyaline cartilage, fibrous connective tissue, and fibrocartilage can live and grow for many years, but bone, although it retains its form and appearance, is dead.

Fig. 12-B: Enlargement (× 25) of rectangle *B* in Fig. 12-A.

Fig. 12-C: Enlargement (× 25) of rectangle *C* in Fig. 12-A.

lesion which was produced experimentally occurred in the proper location and only in that location; its dimensions were typical of those found in clinical cases, and the damage was confined to the same structures as those found damaged in clinical cases.

The existence and significance of the force of torsional-rotation impaction were not noted by previous investigators of the etiology of osteochondritis dissecans, although two hinted at it. Kappis [41], who first described osteochondritis dissecans of the talar dome, suggested that the mechanism involved a tangential twisting force, and Roesner [64] described a tangential twisting force as producing the lesions of osteochondritis dissecans of the knee. Neither mentioned the importance of the role played by the collateral ligaments of the joints in producing rotational impaction.

We suggest that it was the failure to recognize and understand the force of torsional impaction that delayed, for so many years, the acceptance of osteochondritis dissecans as a fracture, and that it is this force which unobtrusively produced these small fractures. As previously mentioned, the lack of pain in the Stage One lesions was due to lack of sensory nerves at the fracture site.

Thus, two of the three objections to the inclusion of osteochondritis dissecans

as a fracture have been overcome. The third, the lack of healing after so-called reasonable immobilization, will be discussed under the heading of pathology.

Pathology

Once the osteochondral fracture has occurred—whether lateral or medial, Stage One or Stage Four—it can heal like other fractures, but only if it is reduced and immobilized until bone union has taken place.

In the Stage Four lesion the displaced chip cannot heal unless the fragment

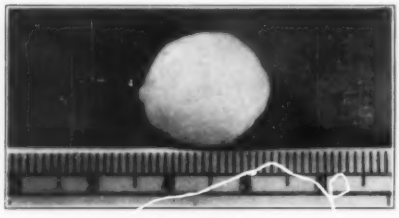

FIG. 13-A

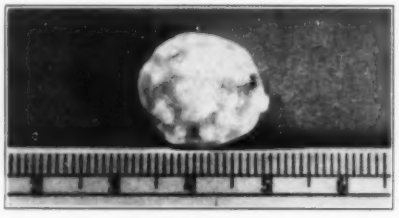

FIG. 13-B

Fig. 13-A: Articular surface of an osteocartilaginous loose body removed from the knee of a man, thirty-two years old. At operation the fragment was lying free in the intercondylar notch, with no attachment to the surrounding tissues. The crater from which it had taken origin on the inferior and lateral aspect of the medial condyle was filled with firm fibrocartilage. Note the smooth normal hyaline cartilage.

Fig. 13-B: Fracture surface of the loose fragment. Note gross appearance of finely nodular cartilage.

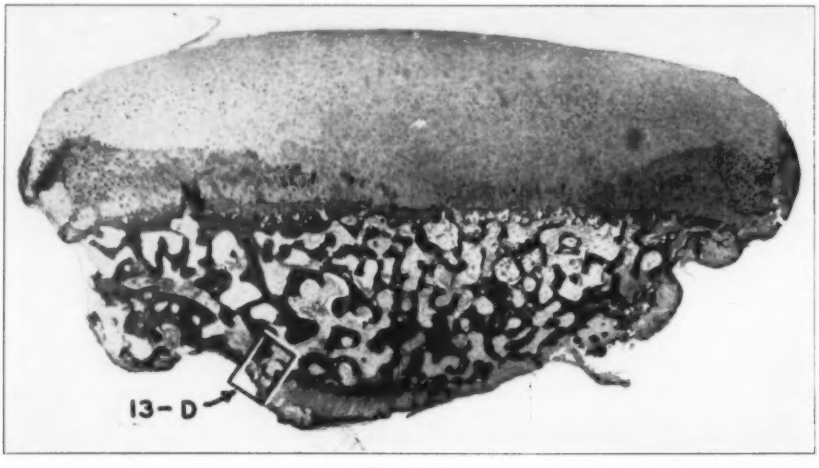

FIG. 13-C

Photomicrograph (× 13) of cross section of osteocartilaginous body. The articular surface (top) is composed of normal hyaline cartilage containing living chondrocytes. In the bone the lacunae are empty and there are no osteoblasts. Fibrous tissue has overgrown the rounded edges of the fragment, covering the fracture surface and penetrating the medullary spaces. About the edges and on the fracture surface the connective tissue has undergone metaplasia to fibrocartilage.

is replaced in its bed. In the other types of fractures—Stages One, Two, and Three—the blood supply of the compressed or avulsed fragment has been cut off at the fracture line. The process of healing can take place only by the growth of capillaries from the parent talus across the fracture line. The bone fragment remains in a condition of avascular, aseptic necrosis until the arrival of a new blood supply to nourish it and to bring scavenger cells to destroy the dead bone.

The cartilaginous portion of the fragment, on the other hand, continues to

live because of its low metabolic requirements. Deprivation of blood supply to the fragment is of no consequence to the cartilage, whose cells receive their nutrition from the synovial fluid. Those cells may grow peripherally about a loose fragment, ultimately investing the trabecular surface with cartilage (Figs. 12-A through 13-D).

Healing of the undisplaced fractures has been reported to occur. Cameron [14] noted in his case report, that it required seven and one-half months for the fracture to show roentgenographic evidence of the establishment of union. In this case, as in others, the recognition of healing was based on roentgenographic evidence and clinical relief of symptoms; both criteria have been shown to be deceiving at times. There have been no reports of exploration or pathological examination of the reportedly healed lesions.

If immobilization of the fracture line is incomplete in degree or inadequate in duration, motion at the fracture line shears off the ingrowing capillaries, and the reparative process ceases. The attempted invasion of capillaries from the host (talus) apparently is a feeble one which is discouraged easily by motion at the fracture line. There have been no reports to tell how much motion can be tolerated, or for what length of time motion can be permitted before spontaneous healing can no longer take place. When the reparative process ceases, the host surface of the fracture becomes covered with dense fibrous connective tissue that seems to be impenetrable to capillaries. The fragment becomes a dead prisoner in a sterile cell.

FIG. 13-D

Photomicrograph (× 100) of fracture surface (See Fig. 13-C). Note fibrocartilage on fracture surface.

The condition of the imprisoned fragment cannot improve, but there are several possibilities of further insult. Continued weight-bearing, with alternating application and release of pressure against the cartilaginous attachment of the Stage One or Stage Two fragment, may rupture the attachment, converting the fragment to the detached Stage Three or the displaced Stage Four lesion. Pressure may break off smaller fragments from the original fragment or from the margin of the crater. These satellite fragments may then be dispersed by motion to the various recesses of the ankle joint (Fig. 14-C). Where such fragments lie in contact with the synovial membrane they incite irritative reactions and may become attached to it. The frictional effect of the uneven joint surface during exercise presumably increases the temperature of the joint, producing the characteristic dull aching that accompanies synovial irritation [37]. Ultimately, degenerative joint disease supervenes. Instability of the damaged talus within the ankle mortise, caused by deficiencies of the previously torn collateral ligaments, may lead to repetitions of the original sprain. Any or all of these eventualities may follow one fracture.

If the necrotic fragment is removed from the joint and the fibrous coating of the host surface curetted away, a new reparative process may begin. It has been reported that the defect becomes filled with fibrous connective tissue that restores the original shape of the talus and is of sufficient firmness and smoothness to act

FIG. 14-A

FIG. 14-B

Fig. 14-A: Anteroposterior roentgenogram of the right ankle (Case 5). Note that in this projection the medial margin of the talus is shown in profile, whereas the lateral margin is obscured by the superimposed fibula.

Fig. 14-B: Oblique projection (10 degrees of medial rotation) of same ankle as Fig. 14-A. Note how well the talofibular joint and the lateral margin of the lateral border of the talus are visualized.

FIG. 14-C

FIG. 14-D

Fig. 14-C: Oblique roentgenogram of the right ankle (Case 6) sixteen months after injury. The fragment on the lateral margin of the talus appears to be intact. A small spherical loose body seems to lie between the talus and the medial malleolus.

Fig. 14-D: Lateral roentgenogram of the same ankle as Fig. 14-C. There appears to be an osteophyte on the anterior lip of the tibia, but at operation both the loose body and the osteophyte were found to be small pieces of the fractured talus displaced and adherent to the synovial membrane.

as a satisfactory replacement for the missing bone [59]. Burr [13] reported a case in which the roentgenograms of the fracture, made seven years postoperatively, showed the defect to be filled with new bone. This statement was not confirmed by pathological examination.

In summary, the sequence of pathological events in the osteochondral fracture is: (1) occurrence of the fracture, (2) immediate development of avascular, aseptic necrosis of the osseous portion of the fragment while the cartilaginous portion lives, and (3) either union of the fracture by creeping substitution or sequestration and non-union of the fracture with the later complication of osteo arthritis. The progress of creeping substitution is measured in months, not in weeks. Fractures involving even small fragments cannot reasonably be expected to heal with the rapidity displayed by other fragments of the same size having intact blood supply.

Symptomatology

There are no symptoms that are pathognomonic of transchondral fractures of the ankle. We could find no record of the diagnosis having been made on the basis of history and physical examination alone. New, or acute, fractures usually were diagnosed clinically as sprains. Chronic cases were diagnosed as arthritis.

Acute phase: The symptoms of new fractures are those of inversion sprain: pain in the torn collateral ligaments, swelling of the ankle, ecchymosis, and limitation of motion.

Duration of acute phase: The duration of symptoms of the acute phase varied

from several weeks to several months, governed by the severity of the accompanying sprain [1,7,18].

Atraumatic acute phase: The only new cases in which no diagnosis of sprain was made were those in which a Stage One lesion occurred without accompanying tear of the collateral ligaments. In these cases of the so-called atraumatic osteochondritis dissecans, the patients did not seek medical attention until the symptoms of the late, or chronic stage became manifest.

Chronic phase: The chronic phase begins with the subsidence of symptoms of sprain. The severity of symptoms depends on the degree of healing of the torn ligaments and on the extent of the fracture. The symptoms of the chronic phase in Stage One, Stage Two, and Stage Three fractures are those of osteo-arthritis (degenerative arthritis, traumatic arthritis, and arthrosis): freedom of pain when the joint is at rest, stiffness, crepitus, aching, swelling, and limitation of motion during and after exercise. In Stage Four fractures locking occurs occasionally in addition to the symptoms of osteo-arthritis and, at times, the symptoms of incomplete healing of the collateral ligaments: weak ankle, instability, pain on inversion of the ankle, and a tendency to recurrence of sprains.

The severity of symptoms of the chronic phase is subject to unexplained variations. Some patients report freedom from symptoms for long periods of time between recurrences (Cases 13, 17, and 18.)

Duration of chronic phase: The duration of symptoms of the chronic phase appears to be indefinite unless surgical intervention occurs. The longest duration of symptoms in a case reported heretofore was nineteen years [65]. In the patient in Case 18 the duration, from childhood until the age of thirty-three, apparently was greater. The longest duration proved by roentgenograms was that of a case first reported by Hellström and Östling [35], with follow-up by Aronsson [2] of a physician, twenty-four years old, who sprained his left ankle. Roentgenograms were reported as showing no injury. One year later, while pursuing a bus, the patient felt a sharp pain in the left ankle. At that time roentgenograms showed an area of osteochondritis dissecans of the lateral border of the left talus. The roentgenographic appearance, when the patient was twenty-six years old, was unchanged and at thirty-eight, roentgenograms showed the lesion still not healed. In this case the duration, by history, was fourteen years and, by roentgenograms, thirteen years.

Physicians who treat sprains or arthritis of the ankle should keep in mind the possibility of the presence of a transchondral fracture that has the potentiality, if it is not identified and treated properly, of causing prolonged disability. Roentgenographic examination is obligatory. A denial by the patient of injury or of previous symptoms should not be accepted as correct unless confirmed by thorough questioning (Cases 13 and 18).

Laboratory Findings

There were no significant variations from normal in the findings in blood and urine, and in cytology in cases of transchondral fractures.

Synovial fluid from the affected ankle has been sanguineous in patients operated on within the first few days after injury (Case 1). We have not encountered any reports of analyses of synovial fluid from patients with chronic conditions.

Roentgenographic Diagnosis

Without exception, in the cases reviewed, the correct diagnosis was not made until the roentgenograms were examined. Clinical diagnoses made prior to that time were of either sprains or arthritis. Delay in having roentgenographic examination was responsible for failure of early recognition of the osteochondral lesions. In the present series the patient in Case 6 had symptoms for one year before roentgenograms were made; in Case 8, for thirty-two years; in Case 10, for eight years; in Case 17, for five years; in Case 18, for about twenty years; and in Case

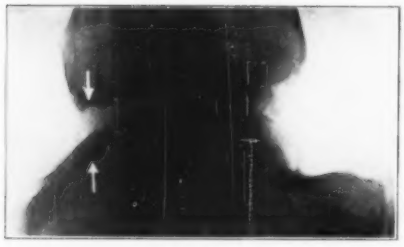

FIG. 15-A

FIG. 15-B

Fig. 15-A: Lateral roentgenogram of the left ankle (Case 14). In this view of a Stage One fracture of the medial border of the talus the arrows indicate indentations of both the posterior lip of the tibial articular surface and of the apposing posterior medial border of the talar dome—evidence of impaction at these points.

Fig. 15-B: Lateral roentgenogram of the left ankle (Case 7). In this view the large displaced fragment of this Stage Four fracture of the medial border of the talus is clearly visualized in the anterior compartment of the ankle joint. Compare this with the anteroposterior view of the fracture shown in Fig. 3-H.

FIG. 15-C

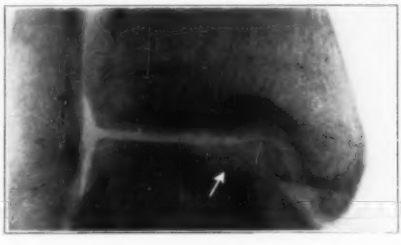

FIG. 15-D

Fig. 15-C: Preoperative roentgenogram of the left ankle (Case 18) showing a Stage Three fracture of the medial border of the talar dome. Note similarity of the appearance of this lesion to that shown in Fig. 3-H where the fragment was displaced into the anterior joint compartment.

Fig. 15-D: Roentgenogram of the same ankle as Fig. 15-C ten months after operation. There is no evidence of bone growth in the crater visible in the roentgenogram.

20, for ten years. The importance of early roentgenographic examination is obvious.

Roentgenologists should be aware of the possibility of the transchondral fracture occurring as a complication of a sprain, either recent or remote, and should be prepared to make an adequate number of exposures before returning a negative report, particularly if symptoms of a sprain are unduly persistent.

A minimum of three views, with the foot in neutral position, should be made: anteroposterior, oblique (10 degrees of medial rotation), and lateral, all centered over the joint line.

The anteroposterior view, the view most commonly made, usually shows the medial margin of the talus clearly, owing to the obliquity of the anteroposterior axis of the talus in relation to the mid-line of the foot, but it obscures the lateral border of the talar dome by superimposition of the lateral malleolus (Fig. 14-A).

The oblique view in 10 degrees of medial rotation opens up the talofibular joint and gives a clearer view of the lateral margin of the talus (Fig. 14-B). This is important in determining the presence of small detached fragments which might be overlooked in preoperative roentgenograms (Fig. 14-C). These fragments must be removed at operation.

The lateral view, although part of the dome is obscured by the overlying malleoli, shows the size of the lesion and indicates whether the lesion should be approached surgically from the anterior or posterior aspect of the ankle (Figs. 1-B and 15-B).

In some cases (Cases 8 and 19) the original roentgenograms were reported as showing no abnormality, but films made several days later showed the lesions clearly. Although it is possible that the roentgenograms made immediately after injury might not have shown the small lesion until more separation of the fragment occurred, it is more likely that the lesion was obscured by the position of the ankle during the first exposures. Additional roentgenograms made with the ankle in more dorsiflexion or more plantar flexion, both oblique and lateral views, should point out the lesion.

The appearance of a sclerotic line about the margin of the crater is suggestive of fibrosis of the fracture crater, implying that the lesion is at least several weeks old and that conservative treatment probably will not be successful.

The roentgenologist should be wary of judging the progress of healing. The fractures in Cases 1 and 17 were reported over a period of months to be healing well, yet later roentgenograms showed the lesions as unchanged from their appearance in earlier films; the errors were introduced by different positioning of the ankle with respect to dorsiflexion and plantar flexion in the anteroposterior views (Figs. 2-A and 2-B). For appraisal of union, the fragment must be shown in profile on top of the dome in all films. If such precautions are taken, union, if present, can be demonstrated by the progressive appearance of trabeculations crossing the fracture line.

Preoperative roentgenograms can give only an approximation of the size of the osteochondral fragment because of the relative radiolucency of the cartilaginous component. The fragment always appears larger at operation than in roentgenographic views.

The postoperative appearance of these lesions will be discussed after the clinical appraisal of postoperative results.

Concomitant Injuries

Transchondral fractures of the talus have been incurred in accidents of various degrees of severity. In some cases the trauma was not recognized as such. Many cases occurred from missteps, falls, and jumps, and some as the result of vehicular accidents.

As might be expected, in the majority of cases there were no concomitant injuries, unless the accompanying sprain is counted as such. Fractures of the lateral malleolus occurred in five patients, of the medial malleolus in four, of the body of the talus in four, and of the calcaneus in one; all within the affected foot. Other and more distant fractures were reported following more serious injuries (Case 6, present series).

Conservative Treatment

The various methods of conservative treatment employed were not described adequately in most of the reports reviewed. The number of weeks of treatment was rarely recorded. The failure to record that information has deprived the medical profession of one of the most valuable items of information that could be learned about these lesions: the length of time necessary for complete immobilization of the ankle to permit bone union of the fracture in children and in adults. Until such a standard—the minimal time necessary—has been proved it could be said that conservative treatment has not had a fair trial.

Without that information, it has been necessary to assume that conservative treatment, in some form, was continued throughout the period of the duration of symptoms and the follow-up period. This arbitrary extension of the period of conservative treatment may seem to be excessive until the type and intensity of different methods of conservative treatment are considered.

The types of conservative treatment mentioned were: rest, dressings, adhesive-plaster strapping, elastic bandages, braces, special shoes, casts, injections of procaine or hyaluronidase, and physical therapy of many types. Not mentioned,

but certainly a part of conservative management, were: avoidance of use of the part when possible, rest of the part after exercise was unavoidable, and the numerous home remedies which families and friends can suggest for the alleviation of joint pain.

Since these methods do not require constant medical attention, it is within reason to presume that some type of conservative treatment to some degree was followed by each patient throughout the period of his disability until (1) the lesion was cured, (2) the lesion was operated on, or (3) the period of observation ended.

Results of Conservative Treatment

In this paper it was necessary to grade results of treatment as simply as possible to permit comparison between methods of treatment. A result was rated *good* if the author reported freedom from symptoms or if only slightly annoying, but not disabling, symptoms persisted. A *fair* result was credited if the symptoms were somewhat improved, but some measure of disability persisted. The result was graded *poor* if the symptoms continued unchanged, even though periods of freedom from symptoms occurred at times. The requirement of later operation has been taken as *prima facie* evidence of a poor result of earlier treatment.

Conservative treatment was reported in 168 fractures, but the results of treatment were mentioned in only 154. The results were: good in twenty-six fractures (16.9 per cent), fair in thirteen (8.5 per cent) and poor in 115 (74.6 per cent).

Of the 154 fractures, fifty (32.5 per cent) required later operation.

The astonishingly low percentage of good results, particularly in view of the statements by some authors [8,19,29] that these fractures are minor in character, not serious, and require only a few weeks of immobilization of the ankle, shows that the fractures have been underestimated and inadequately treated.

The popularity of conservative treatment has not been justified in view of the poor results obtained. It must be noted that conservative treatment with proper immobilization should give good results if employed promptly and long enough, but the results reported up to the present indicate that in most cases the degree and the duration of immobilization have not been adequate. One factor that has contributed to the poor results of conservative treatment has been the occurrence of so-called atraumatic osteochondritis dissecans in which the lack of symptoms during the acute phase precluded treatment until the development of late (arthritic) symptoms, when conservative treatment no longer could be expected to improve conditions.

Indications for Surgical Treatment

Seventy-three operations were performed on seventy patients. Of these, the indications for operation were apparent in fifty-seven. Five who were operated on had new or very recent injuries (8.8 per cent). Failure of previous conservative treatment led to fifty operations (87.7 per cent). Failure of previous surgical treatment, due to incomplete removal of fragments, necessitated reoperation in two cases (3.5 per cent).

Types of Surgical Treatment

Three types of operations were recorded in seventy-two fractures: arthrotomy for inspection only, removal of fragment, and replacement of fragment.

Arthrotomies without definitive surgery were reported to have been done when the lesions shown in roentgenograms could not be found at operation owing to the overlying cartilage's being intact. Four such arthrotomies were performed (5.5 per cent) in the seventy-two operations.

Removal of the fragment was the operation most often performed. It was used in sixty-five cases (90.3) per cent.

Replacement of the fragment was performed in three cases (4.2 per cent).

Results of Surgical Treatment

The results of operation, on a basis of clinical appraisal, were reported in sixty-two cases.

Arthrotomy, which should be regarded as having done nothing to improve the condition of the lesion and as having added the trauma of surgery, was judged as giving fair results in one case and poor in two. Results were not reported in one case.

Replacement of the fragment, employed in Stage Four lesions, produced good results in all three cases.

Removal of the chip, with results recorded in fifty-six operations, was reported as good in forty-seven cases (84 per cent), fair in six cases (10.7 per cent), and poor in three cases (5.3 per cent). Of the three cases with poor results, two were attributed to incomplete removal of fragments at operation. Both were reoperated with subsequent good results (included in the forty-seven good results just mentioned). The single remaining poor result of operation was not discussed by the authors.

Complications of Surgical Treatment

The following complications were reported: non-union in one case; incomplete removal in five cases, two of which required secondary operation; osteotomy of a malleolus, done to obtain adequate exposure, in four cases; osteo-arthritis in three cases; causalgia in two cases; and infection in one.

Results of Treatment in Children

The results of treatment previously given are those reported by the authors for all cases without respect to the age of the patients.

Some authors, writing on osteochondritis dissecans of joints other than the ankle, reported that the lesions heal spontaneously in children under seventeen years of age in a much higher percentage of cases than do similar lesions in adults. Van Demark [74] and Wiberg [78,79] reported cases in which lesions in children's knees healed completely during prolonged conservative treatment, the duration stated to be at least six months, without weight-bearing. Van Demark recommended the following general rules for treatment of lesions of the knee: if the age of the patient is sixteen years or less, immobilize for six months or more; if the age is between sixteen and twenty years, the best method of treatment is uncertain; if the age is more than twenty years, operate to remove the fragment.

In order to compare the methods of treatment and their results in the cases of transchondral fractures of the talar dome reviewed herein with the opinions previously reported concerning lesions of the knee, a review of all the reported cases involving the talus in children was made.

There were no cases reported in children of sixteen years, but in fourteen children of fifteen years or less there were sixteen fractures (two were bilateral). The youngest patient was five years of age; one was nine; and the rest were eleven to fifteen.

Results of Conservative Treatment in Children

Conservative treatment was employed in fifteen of these fractures. The results were: good in three cases, fair in one, and poor in eleven. Of these fifteen conservatively treated cases, seven patients were operated on at a later date.

Results of Surgical Treatment in Children

Nine operations were performed on eight children. One had a new injury. Seven had had previous conservative treatment. One of the latter group had a second operation because of incomplete removal of fragments at the first operation. The results reported were: good in eight and poor in one (Table V).

TABLE IV

RESULTS OF TREATMENT AND DURATION OF FOLLOW-UP IN ALL REPORTED CASES OF TRANSCHONDRAL FRACTURES

Results of Treatment	Conservative Treatment Follow-up (*Years*) 0 to 1	1 to 2	2 to 5	5 and Over	No. of Cases	Per Cent of Total	Surgical Treatment Follow-up (*Years*) 0 to 1	1 to 2	2 to 5	5 and Over	No. of Cases	Per Cent of Total
Good	8	2	8	8	26	17.4	23	5	11	5	44	78.6
Fair	2	6	2	3	13	8.7	3	2	2	0	7	12.5
Poor	41	21	24	24	110	73.9	2	1	2	0	5	8.9
Totals	51	29	34	35	149	100.0	28	8	14	5	56*	100.0

* Fifty of the surgical cases had had failure of previous conservative treatment.

TABLE V

RESULTS OF TREATMENT AND DURATION OF FOLLOW-UP OF TRANSCHONDRAL FRACTURES IN CHILDREN

Results of Treatment	Conservative Treatment Follow-up (*Years*) Not Given	0 to 1	1 to 2	2 to 5	5 and Over	No. of Cases	Per Cent of Total	Surgical Treatment Follow-up (*Years*) Not Given	0 to 1	1 to 2	2 to 5	5 and Over	No. of Cases	Per Cent of Total
Good	0	1	0	1	1	3	20.0	1	2	1	2	2†	8*	88.0
Fair	0	0	1	0	0	1	6.7	0	0	0	0	0	0	0.0
Poor	1	2	2	1	5	11	73.3	0	1†	0	0	0	1	12.0
Totals	1	3	3	2	6	15	100.0	1	3	1	2	2	9	100.0

* Seven surgical cases had had failure of previous conservative treatment.
† One patient operated on twice; poor results after incomplete removal at first operation; good results after second.

Summary of Treatment in Children

On the basis of the results of treatment in children it would appear that the conservative treatment of ankle lesions was not so successful as that reported by Van Demark and Wiberg in knee lesions. Perhaps the duration of immobilization for those children in whom the talus was involved was not so long as that recommended by Van Demark; the duration was not recorded, although follow-up was listed in seven cases as varying from five months to twelve years. All of these patients had poor results.

In brief, the methods of treatment in children under sixteen years of age were productive of the same results as in adults; conservative treatment gave poor results, operative treatment gave good results.

Duration of Follow-up

The duration of the follow-up periods of treatment of these fractures was adequate to permit evaluation of the various methods of treatment employed. The periods of follow-up for the conservatively treated cases were approximately the same as for those treated surgically (Table IV).

The appraisal of results of treatment by clinical methods within a few months after treatment is open to challenge, no matter what method of treatment has been employed. All methods of treatment had in common some period of immobilization which would give at least temporary benefit; this in turn, if appraised too early, might be mistaken for a good final result. Furthermore, since some patients experienced long periods of complete freedom from symptoms only to have later recurrences, appraisal too early again might give false information. It is suggested that a period of two years of follow-up be designated as the minimal acceptable duration in future reports.

Roentgenographic Evaluation of Treatment

After all types of treatment, except that of removal of the fragment, the interpretation of follow-up roentgenograms does not present any problems not already encountered in the preliminary roentgenographic diagnosis of the lesion.

If the fragment has been removed, little information can be expected from postoperative roentgenograms as far as the progress of the crater is concerned (Table I). Filling of the crater by fibrous connective tissue could not be expected to show in the roentgenograms. Burr [13] reported that the crater filled with bone in seven years.

The greatest value of the postoperative roentgenographic examination lies in the identification of residual small fragments as the cause of continuing symptoms.

LESIONS OF OTHER JOINTS

The lesions discussed in this paper, with the exception of the case described in Figure 13, pertained only to the talus. Although we have not performed anatomical experiments on joints other than the ankle, we believe that the conclusions reached in this study concerning the etiological mechanism and methods of treatment apply with equal accuracy to the lesions of osteochondritis dissecans that are found in other joints—particularly that of the medial condyle of the knee—and to fractures of the radial head.

SUMMARY

Detached osteochondral fragments from the dome of the talus have been described in orthopaedic publications by a variety of confusing terms. A clarification of this conflicting terminology is suggested.

We believe that the traumatic etiology of the lesion has been confirmed both clinically and experimentally. The problems of clinical and roentgenographic diag-

nosis are discussed and illustrated by case histories. Various forms of treatment are evaluated, and the prognosis appraised.

NOTE: We express our gratitude to the attending surgeons who permitted the incorporation in this report of the histories and roentgenograms of their cases: Dr. G. W. Bennett, Case 21; Dr. Robert R. Crawford, Case 7; Dr. William M. Davis, Case 3; Dr. Z. B. Friedenberg, Case 18; Dr. M. C. Goodman, Cases 14, 17, and 20; Dr. W. H. Hull, Case 12; Dr. A. W. Humphries, Cases 8 and 16; Dr. M. C. Kolczun, Case 5; Dr. J. T. Nicholson, Case 15; Dr. George S. Phalen, Cases 2, 6, 13, and 19; Dr. J. M. Strong and Dr. R. C. Novatny, Case 4; Dr. J. M. Strong and Dr. G. W. Bennett, Case 11; and Dr. J. F. Whitman, Case 10.

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Gastrocnemius-Muscle Recession (Silfverskiold Operation) for Spastic Equinus Deformity in Cerebral Palsy*

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This presentation comprises an eleven-year study of cerebral palsy in 525 children (not including primary speech and hearing cases) at the Meeting Street School in Providence, Rhode Island, from 1947 to 1958. Two hundred and fifty-three operative procedures on the lower extremities were performed in eighty-eight patients. Seventeen per cent of the patients, therefore, were considered candidates for such surgery.

The function and philosophy of the Meeting Street School must be explained at the outset. The condition of each child in the Meeting Street School is evaluated, and his progress followed by a pediatrician (who is the Medical Director), a neurosurgeon, an orthopaedic surgeon, a psychologist, a social worker, a physiotherapist, an occupational therapist, and a speech therapist. Consultation by other specialists is readily available as the specific indication arises. Frequent group staff meetings are held for round table discussion of the individual cases.

In this integrated organization, orthopaedic surgery is merely one facet in the comprehensive treatment of cerebral palsy in children. All patients are studied thoroughly for at least six months prior to the consideration of prospective surgical treatment. During this period they receive intensive physiotherapy and are fitted with braces and night splints when indicated.

The role of orthopaedic surgery in the treatment of deformities of the lower extremities is primarily that of helping the patient to attain the ability to walk independently and secondarily that of improving gait and preventing the development of increasing deformities in the patients who have failed to respond to conservative therapy.

HISTORICAL SURVEY OF SPASTIC EQUINUS CONTRACTURE

Spastic equinus contracture is the most common deformity of the lower extremity encountered in cerebral palsied children. For centuries tenotomy of the Achilles tendon has been performed to correct equinus deformity. The results of this procedure in cerebral palsy have been disappointing, since Achilles-tendon lengthening does not overcome the hyperstimulation of the gastrocnemius muscle by the tibial motor nerve supply. Significant recurrence of the equinus deformity has been reported by a number of authors [4,8,10].

In 1913 Vulpius and Stoffel described an operative procedure for equinus deformity in which the broad gastrocnemius tendon was sectioned transversely just below the middle of the leg posteriorly and the soleus tendon was cut transversely, leaving the underlying muscle fibers intact. Then, by forcible dorsiflexion of the foot, the soleus muscle fibers were passively stretched without losing the continuity of the soleus muscle.

In 1924, Silfverskiold made an important contribution to the analysis and treatment of spastic equinus contracture. He divided this deformity into two significant groups:

1. Equinus that is essentially inflexible with the knee fully extended, but

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capable of being corrected passively with relative ease when the knee is flexed to a right angle;

2. Persistent equinus, whether the knee is extended or flexed.

In the first type he reasoned that the gastrocnemius muscle was the primary deforming factor, since, when it was relaxed by flexing the knee, the equinus contracture was overcome. Silfverskiold advocated lowering, or recession, of the gastrocnemius muscle heads below the level of the knee joint, converting a two-joint muscle into a one-joint muscle. In effect, he converted the gastrocnemius into a second soleus muscle which now had its origin below the knee joint. In addition, he advised partial section of the tibial motor nerves to the gastrocnemius muscle, to reduce its power further. The soleus muscle remained unaffected and could be used for push-off from the fore part of the foot in walking.

In the second type in which the entire calf-muscle mechanism (gastrocnemius and soleus) is contracted, lengthening of the Achilles tendon is indicated.

The second type is less commonly encountered, especially in younger children. In our total series, Achilles-tendon lengthening was done in only four children three, four, eight, and eleven years of age with fixed contracture of both the soleus and the gastrocnemius muscles.

Green and McDermott, in 1942, in a review of operative procedures in cerebral palsy, listed lengthening of the upper end of the gastrocnemius muscle for equinus deformity in fifteen instances and noted that in four of these neurectomy to one head of the gastrocnemius was done also.

In 1950, Strayer described transverse section of the gastrocnemius tendon at its junction to the conjoined gastrocnemius soleus tendon, and resuture of the retracted gastrocnemius tendon with the foot dorsiflexed to 90 degrees. He concluded that the beneficial result in the cases reported was due to alteration of the proprioceptive reflexes in the central nervous system, with modification of the stretch reflexes in the extremity.

In a subsequent article in October 1958, Strayer reviewed his results in twenty-three cases, with good results in eighteen, his cases having been followed three to ten years.

Baker, in 1954, reported recession of the gastrocnemius heads from the femoral condyles to below the knee joint. Neurectomy was not performed in his cases.

We have performed the Silfverskiold gastrocnemius-recession operation for spastic equinus deformity since 1948, and wish to report our results to the present.

In 1957, we presented [7] an eight-year study of the results of the surgical treatment of cerebral palsy in children at the Meeting Street School up to 1955. At that time, we had done seventy-three Silfverskiold operations, with three recurrences of equinus contracture.

TECHNIQUE OF SILFVERSKIOLD OPERATION

The operative technique has been described by us previously [7] but will be reviewed here. The Silfverskiold operation is done with the patient lying prone, under general anesthesia, and is always performed under tourniquet control (mid-thigh). In cases of bilateral involvement the knees are preferably operated on simultaneously by two orthopaedic surgeons who are familiar with the procedure. In this manner the operating time and the duration of the anesthesia are kept to a minimum. When there are adduction contractures of the hips also, the hip-release procedures are performed first. At the conclusion of the hip surgery, the patient is turned over into the prone position; tourniquets are applied to the thighs, the knees prepared and draped, and the Silfverskiold operation performed on both knees. During the past seven years, in bilateral cases, we have routinely operated on both hips and knees in a single operative session, when indicated. The total operative time for the bilateral hip and knee releases has ranged between one hour and one hour and fifteen minutes, and an unnecessary second operation has been avoided.

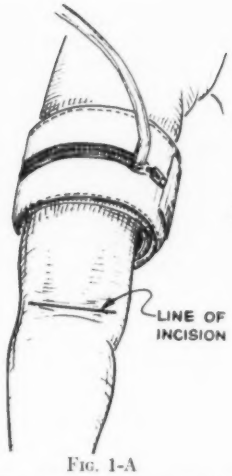

FIG. 1-A

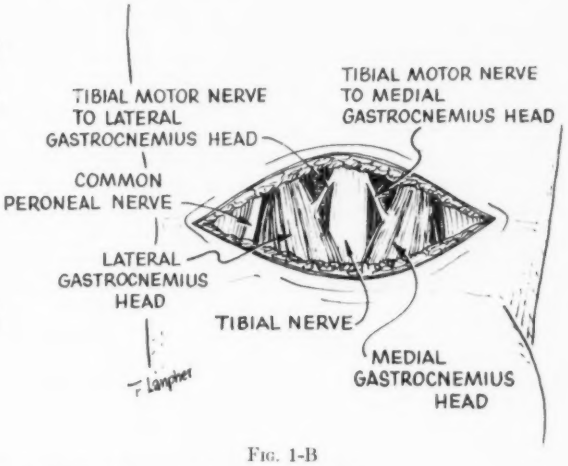

FIG. 1-B

Fig. 1-A: Transverse incision in popliteal crease. A tourniquet is used.
Fig. 1-B: Significant structures exposed in the operative field.

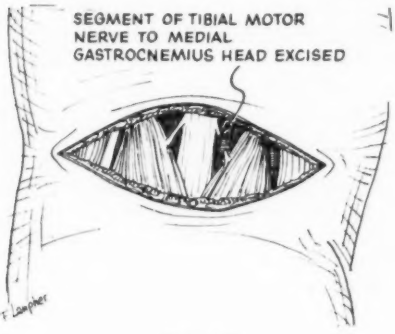

FIG. 1-C

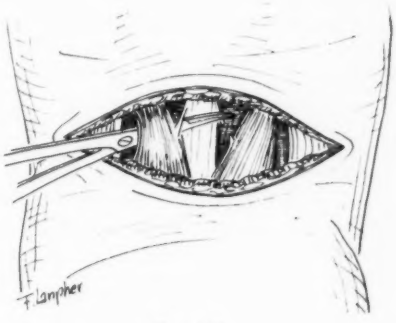

FIG. 1-D

Fig. 1-C: Neurectomy of motor nerve to medial gastrocnemius head.
Fig. 1-D: Sectioning of lateral gastrocnemius head from femoral condyle. Motor nerve to lateral gastrocnemius head is intact.

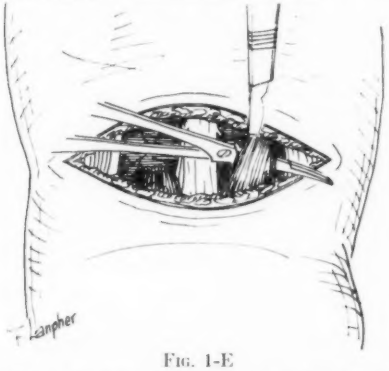

FIG. 1-E

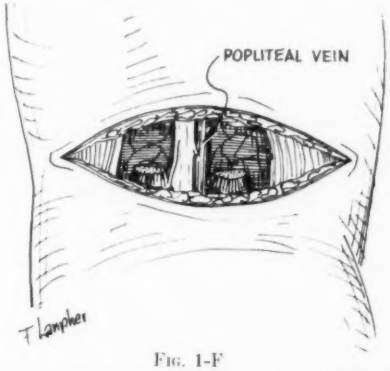

FIG. 1-F

Fig. 1-E: Lateral gastrocnemius head has been recessed below level of the knee. Medial gastrocnemius head about to be sectioned.
Fig. 1-F: Both gastrocnemius heads have been recessed distally. The popliteal vein is immediately under the tibial nerve.

The incision is a transverse one in the popliteal crease, which minimizes any tendency to the formation of a thickened scar (Figs. 1-A through 1-G). The incision is deepened transversely through the deep fascia, and the two heads of the gastrocnemius muscle are exposed. Except for dividing and ligating the saphena parva vein, no ligatures are ordinarily needed. The tibial nerve is identified, and the motor branches coursing obliquely downward into the gastrocnemius heads are isolated and identified by gentle pinching. One or two branches will appear on either side of the tibial nerve, running into the medial or lateral head of the gastrocnemius muscle. One-half or more of the motor nerve branches to the gastrocnemius are sectioned, and a small segment of the nerve is excised. In isolating the tibial nerve and identifying its motor branches to the gastrocnemius heads, the utmost care is necessary, since the popliteal vein lies immediately underneath the tibial nerve.

Each head of the gastrocnemius muscle is picked up on a curved Kelly clamp and cut transversely at its proximal attachment, thus freeing it from the posterior aspect of the femoral condyles. Then with a gauze sponge the two muscle heads are wiped down by blunt dissection until they are below the level of the knee joint. In sectioning the lateral head of the gastrocnemius one must bear in mind that the peroneal nerve is immediately adjacent and must be protected from injury.

Closure of the wound is effected by the use of interrupted chromic catgut sutures for the deep fascia while the skin and subcutaneous tissues are closed as a single layer with continuous ethalon or silk sutures to save time. Wound healing in this region has routinely been good. Padded plaster-of-Paris casts are applied from the toes to the groin with the feet dorsiflexed to 10 degrees above a right angle and the knees fully extended.

POSTOPERATIVE CARE

It is essential to cut out a window in the casts over the apex of the heels the day after operation. Early in the series pressure sores on the heels developed despite heavy padding in a number of children without any overt pain. Since the heel windows have been cut out routinely, this complication has been eliminated. The casts are worn for six weeks and are then discontinued. Physiotherapy thereafter is carried out persistently at the Meeting Street School.

Children in the nursery school (three to six years of age) are seen twice weekly at the Meeting Street School, while children over six years of age are seen every three weeks. Participation in gross motor activities for the appropriate age level is stressed, including tricycle riding, roller-skating, and so forth. A regular exercise program is carried on at home. This includes passive, active-assisted, and resistance exercises as indicated for all the hip and knee muscles, for the dorsiflexors of the ankle, and for the extensors and flexors of the toes. Deep knee bends with the feet flat on the floor are practiced, and four months after operation bouncing and jumping with both feet are started. For the patients living at home transportation is provided by the Meeting Street School if need be.

It cannot be too strongly emphasized that the postoperative care must be intelligent, conscientious, and sustained. The postoperative treatment spells the success or failure of carefully performed surgery.

We have not employed braces or night splints postoperatively, but would consider them desirable if intensive supervision of the postoperative care could not be assured.

DISCUSSION OF CASES AND RESULTS

We have performed the Silfverskiold gastrocnemius-recession operation in 110 instances, from 1947 to 1958, and consider it the most reliable soft-tissue operation for spastic equinus deformity, if the indications are properly met.

As stated previously, it allows the conversion of the two-joint gastrocnemius muscle to a one-joint muscle, thus eliminating the primary deforming factor in the

type of equinus contracture which is passively correctable with the knee flexed. Of equal importance is selective neurectomy of the recessed gastrocnemius muscle, which results in permanent partial weakening of this muscle and eliminates the hyperstimulation which has a tendency to reproduce the spastic equinus contracture.

Originally, all the motor nerve branches to the gastrocnemius heads were sectioned and resulted in a poor push-off during walking in eight children (six bilateral cases, two unilateral cases: fourteen operations). During the past two years, we have routinely sectioned the motor nerves to only one gastrocnemius head, with retention of partial function of the recessed gastrocnemius muscle. In twenty-one operations performed in this manner, preliminary studies to date indicate that the resulting function is definitely better.

Forty-four children had bilateral operations (eighty-eight gastrocnemius recessions), and twenty-two had unilateral operations. In eighty-nine of these 110 operations adductor release at the hip was performed at the same operative session. There was no essential difference in frequency of involvement of the left or right sides in the unilateral cases.

Seventeen children had hemiplegic involvement; ten, diplegia; three, triplegia; and thirty-six, quadriplegia. No monoplegia was observed in our series. Eight children presented an associated athetosis, and these were all in the quadriplegic children.

The ability or inability of the child to stand or walk, irrespective of age, is not considered a criterion of eligibility for corrective surgery for the equinus deformity.

Thirty-one of the children in this series were unable to walk at time of surgery, while twelve could walk with support (crutches) and twenty-three could walk independently.

Of the thirty-one children unable to walk at the time of surgery, twenty were four years of age or older. Four years of age has been taken as a rough and arbitrary base line for determining inability to walk. However, there are many factors such as degree of body involvement, general muscle strength, intelligence quotient, degree of spasticity, and associated athetosis which must be taken into consideration in the evaluation of each case. Follow-up study of these twenty patients is presented in Table I.

The postoperative follow-up period has ranged from one to nine years with an average follow-up of four years. The results have been followed for five years or longer after forty-four operations, for two to five years after thirty-four operations, and for less than two years after thirty-two procedures.

Actually, until the child has reached the age of thirteen or fourteen, when full growth of the lower extremity has been practically attained, none of the cases can be considered as end results in the evaluation of correction of spastic equinus.

Five failures (recurrence of equinus) have been observed in our present series of 110 gastrocnemius-recession operations. All of these were apparent within two years after operation. In each instance failure was due to weakness of the dorsiflexor muscles of the foot which was not properly evaluated before operation. These failures do not reflect on the merits of the Silfverskiold operation.

Phelps emphasized that in any case of spastic paralysis three types of muscles may be observed: (1) spastic, (2) normal, and (3) flaccid and paralyzed. A careful muscle survey should be made, and in some instances may have to be repeated to obtain an adequate analysis of the functional muscle relationships.

The evaluation of the dorsiflexor power may not be easy. In the very young child, testing may be done by seating the child with the knee flexed to a right angle thus decreasing the power of the gastrocnemius. The sole of the foot is then stroked repeatedly to produce an extensor response. If adequate dorsiflexor power is present, the response should be prompt, with appreciable force.

If the child is old enough to cooperate he is asked to dorsiflex the foot and

TABLE I

Case		Age at Operation	Time after Operation when Walking Was Begun
1. Walking independently (7 patients)	B.C.	6	1 yr.
	B.G.	4	2 yrs.
	R.L.	11	1 yr.
	L.M.	5	1 yr.
	S.M.	4½	2 yrs.
	C.T.	6	3 yrs.
	J.G.	4	4 yrs.
2. Walking independently with crutches (4 patients)	A.M.	4½	2 yrs.
	D.C.	4	2 yrs.
	D.P.	5	1 yr. (Canadian crutches)
	E.S.	4	4 yrs. (marked spastic quadriplegia)
3. Walking, holding handle of carriage (3 patients)	L.B.	5	3 yrs.
	J.T.	4	1½ yrs.
	T.T.	6	3 yrs.
4. Not walking (6 patients)	M.B.	9	3 yrs. follow-up
	M.C.	6	3 yrs. follow-up
	M.F.	13	5 yrs. follow-up
	L.G.	5	1½ yrs. follow-up
	P.M.	4	6 yrs. follow-up
	S.P.	5	6 yrs. follow-up

TABLE II

INTELLIGENCE LEVELS IN CHILDREN OPERATED UPON

I. Q.	No. of Children
Above 110	4
90-100	12
70-90	35
Below 70	15
Total	66

the muscle strength is tested in the usual fashion. The test should be done repeatedly to make sure that the results are accurate. Electrical stimulation has not been employed, but this might well be a valuable adjunct for muscle testing.

It is essential that fairly good power be present in the dorsiflexor muscles of the foot, otherwise the gastrocnemius-recession operation will produce only a temporary correction. If the dorsiflexor muscles are quite weak, foot-drop with resultant equinus deformity will result despite calf-muscle release, nerve section, or Achilles-tendon lengthening. In such cases, the treatment of choice is passive maintenance of the foot at a right angle with a drop-foot brace until a satisfactory age is reached for stabilization of the foot by arthrodesis. A posterior bone-block operation of the Gill or Campbell type may be carried out, or a Lambrinudi foot stabilization may be indicated.

We do not believe that one can make an accurate graded evaluation of the result of an operative procedure for spastic equinus deformity other than to determine whether the equinus deformity has remained corrected or has recurred.

TABLE III

SPASTIC EQUINUS DEFORMITY TREATED BY GASTROCNEMIUS-RECESSION OPERATION

Age of Operation	No. of Operations	Postoperative Follow-up Period		
		0 to 2 yrs.	2 to 5 yrs.	5 yrs.+
19 mos.	1			1
2 yrs.	5	2	2	1
2½ yrs.	2	2		
3 yrs.	15	5	2	8
3½ yrs.	7	2		5
4 yrs.	22	5	8	9
5 yrs.	14	4	6	4
6 yrs.	25	6	10	9
7 yrs.	6	1	2	3
8 yrs.	5	1	4	
9 yrs.	3	2		1
10 yrs.	1			1
11 yrs.	0			
12 yrs.	0			
13 yrs.	4	2		2
	110	32	34	44

In children with cerebral palsy many factors, which cannot be dissociated from the foot and leg mechanism, play an integral role in determining the results of a surgical procedure. These factors include hip contractures, strength or weakness of the pelvifemoral and trunk muscles, involvement and coordination of the upper extremities, intelligence, and balancing reflex. It is interesting to note that in eighty-nine operations in our series threre was also release of hip adductors performed at the same operative session.

Intelligence

Care was taken to evaluate the prospective candidates for operation from the standpoint of gross intelligence. At least a fair degree of intelligence, that is, an intelligence quotient of 50 or more and demonstrated ability to cooperate were considered prerequisites (Table II). If the consensus of the medical staff of the Meeting Street School was that ambulation in the future could not be expected because of severe mental retardation, then surgery (except for occasional hip release to facilitate nursing care) was considered contra-indicated.

Age at Operation

Given well defined spastic deformities of the lower extremities, the earlier the age at which definitive surgical intervention is undertaken the better to prevent disabling secondary contractures of joint capsules and neurovascular structures.

Early surgery eliminates the physiological overstimulation of the reflex pathways from the calf-muscle group, as discussed by Strayer, and decreases the overflow into other limb and trunk muscles.

On the basis of the age at which the developmental progress and abnormal neuromotor patterns seem fairly well established, the optimal age for operation seems to be about three years. Only twenty-three of the operations in this series were performed at three years of age or younger because many of the patients came under the care of the Meeting Street School at a later age. A period of conservative treatment (of at least six months) was always carried out in these children until failure of conservative therapy was clearly established. The average age at the time of operation was five years with a range of nineteen months to thirteen years. The youngest, a boy, nineteen months of age, presented a markedly spastic unilateral equinus deformity, which failed completely to benefit from conservative treatment, but which responded well to gastrocnemius recession and section of the tibial motor nerve to the gastrocnemius (Table III).

Time in Hospital

The average youngster was admitted to the hospital the day prior to the operation and was discharged three days after its performance. The postoperative course was supervised closely by Dr. Denhoff (Medical Director of the Meeting Street School) and his staff. Morbidity was infrequent, consisting primarily in involvement of the respiratory tract in children who could not cough or swallow adequately. There were no fatalities.

Surgical results for the lower extremities can be properly evaluated only in their dynamic walking pattern; still photographs have little value in cerebral palsy. It has been routine at the Meeting Street School to make a motion-picture film of each child before and after operation at periodic intervals. We have noted that clinical impressions are often misleading in retrospect, and muscle charts alone do not yield an accurate picture of the walking pattern. Such motion-picture records are of great value in the study and evaluation of the operative procedures and the child's over-all progress.

SUMMARY

Spastic equinus contracture is the most common deformity of the lower extremity in cerebral palsied children. In 1924, Silfverskiold divided spastic equinus contracture into two groups, one of which is passively correctable by flexion of the knee to a right angle. The gastrocnemius function is the primary deforming factor in this group and is the most frequently encountered in children.

The Silfverskiold operation, consisting in gastrocnemius-muscle recession with partial neurectomy, is an effective and reliable procedure for spastic equinus deformity when the proper indications are present.

One hundred and ten gastrocnemius-recession operations were performed by the authors between 1947 and 1958 with five failures (recurrence of equinus deformity).

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The Central-Graft Operation for Fusion of Tuberculous Knees, Ankles, and Elbows

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The purpose of this paper is to present our experience in treating tuberculous joints by means of the central-graft operation. This technique of fusing a joint was brought to our attention by Hatt in 1940, and has been so successful in our hands that we believe it is worth reporting. One of the distinct advantages of the procedure, as stated by Hatt, is that a bone graft can be driven across the central portion of a joint, thus bridging and locking its articular surfaces, although not interfering with growth of the bone. Such a procedure is especially applicable to children whose joints may require fusion, since central grafts do

FIG. 1

Roentgenogram showing tuberculosis of the left knee four years after the central-graft operation for fusion. Note that the epiphyseal lines are open and appear the same as on the knee not operated on. When the patient was fifteen years old there was shortening of half an inch.

not arrest bone growth while they are stimulating bone union (Fig. 1). In no instance have we found that a centrally placed bone graft has caused local cessation of bone growth in a child, although some shortening of the limbs, on which this procedure was performed, has occurred in many of the cases. Our experience, therefore, corresponds with that of Hatt.

We have employed the central-graft fusion for tuberculosis of the knee, the ankle, and the elbow with the following results.

TUBERCULOSIS OF THE KNEE

As in the case of all patients suffering from tuberculosis of bone, those with

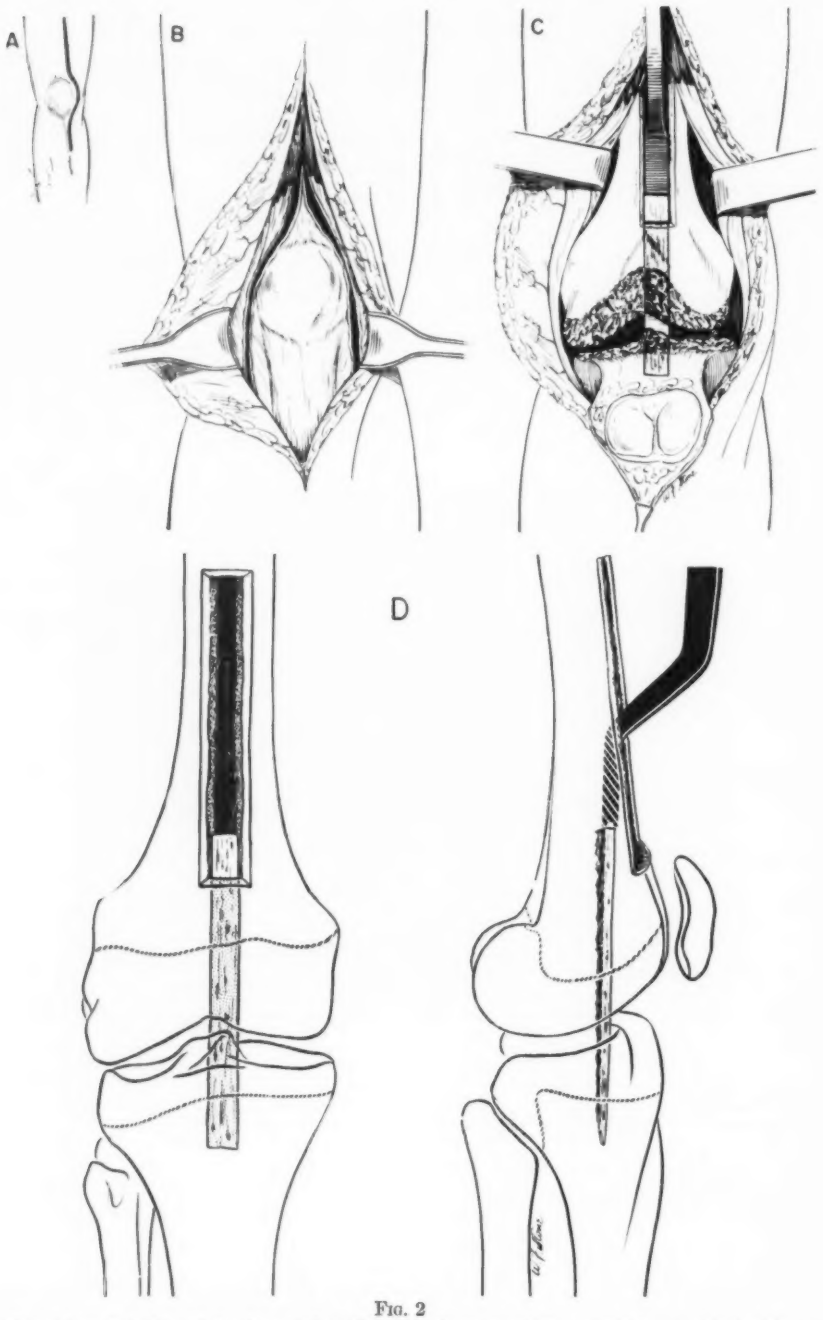

FIG. 2

Central-graft fusion of the knee joint (Coonse-Adams surgical approach): *A*, skin incision; *B*, double parapatellar incision; *C*, patellar flap turned down, exposing the knee joint; massive femoral-bone graft being driven across the center of the joint, after erasion of the articular cartilage; *D*, central graft being driven into proper position, shown in anteroposterior and lateral views.

knee-joint involvement were treated by a preliminary period of bed rest and antituberculosis drugs. In the cases of knee-joint involvement, plaster-cast immobilization was also used until the joint symptoms were considered to be quiescent. The great majority of patients, however, were operated on before the advent of streptomycin and other antituberculosis drugs. All of the joints fused and both groups of patients—those who did not receive antituberculosis drugs and those who did—seemed to do equally well.

There were thirty-four patients—eighteen males and sixteen females, of whom nineteen were adults and fifteen were children—whose tuberculous knees were treated by central-graft fusion. Of these, ten of the adults had dual central-graft fusions.

Single Central Graft for Knee Fusion

The surgical approach of Coonse and Adams is used (Fig. 2, *A* through 2, *D*) with a pneumatic tourniquet applied to the thigh, and the knee joint is exposed, followed by erasion of the articular cartilage and excision of any diseased synovial membrane.

An autogenous-bone graft, about four inches long and half an inch wide, is removed from the anterior surface of either the upper end of the tibia or the lower end of the femur, care being taken to keep a good inch away from the adjacent epiphyseal line. Whether the tibia or the femur is selected for the donor site

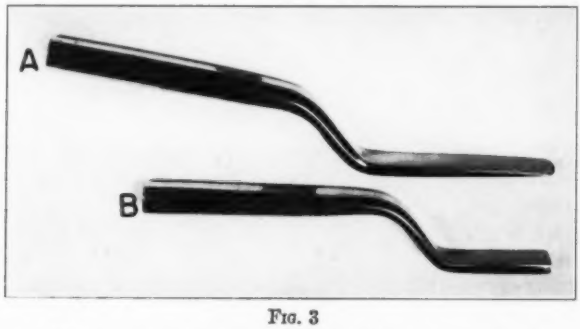

FIG. 3

A, Hatt bayonet osteotome used for making the tunnel; *B*, Hatt bayonet driver or bone set used in placing the graft.

depends on the location of the disease. If, for instance, the tibial articulation is more involved than the femoral, then the graft is removed from the lower part of the femur and driven from above downward across the central portions of the articular surfaces of both the femur and tibia after the method of Hatt.

If, on the other hand, the disease is more extensive in the femur than in the tibia, then the upper portion of the tibia is chosen for the donor site and the graft is driven from below upward through the center of the joint, bridging the two bones and locking them in the desired angle for fusion.

The preparation of the transarticular tunnel through which the bone graft is later driven is greatly facilitated by the employment of Hatt's bayonet osteotomes and driver (Fig. 3); in fact, these instruments are considered to be absolutely essential for this kind of operation.

In adults, the cartilage on the undersurface of the patella is usually removed before closure of the wound, but in children this procedure should never be done because of possible growth disturbance, which is likely to occur if the denuded under surface of the patella comes into contact with the anterior epiphyseal line of the femur and fuses to it. When this occurs (Fig. 4-A), growth of the anterior portion of the lower part of the femur ceases at the site of patellar fusion while the

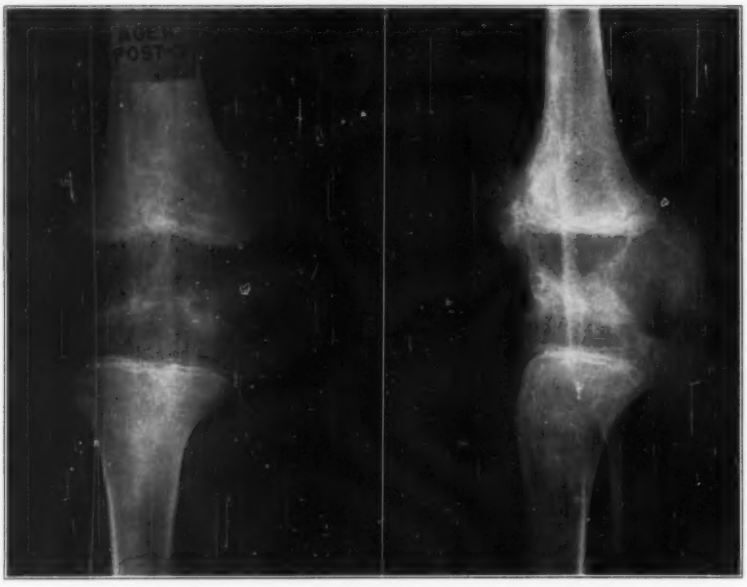

FIG. 4-A

Roentgenogram of tuberculous knee joint, made after central-graft fusion. Note the close contact between the bared undersurface of the patella and the anterior epiphyseal line of the lower end of the femur.

FIG. 4-B

Note that the patella has become fused to the anterior epiphyseal line, causing cessation of growth and resulting in a marked genu recurvatum.

posterior portion continues to grow, producing a serious degree of genu recurvatum (Fig. 4-B). This complication occurred in four of our patients, and corrective osteotomies were required (Figs. 4-C and 4-D). To avoid this danger, either the

FIG. 4-C

Fusion of the patella to the anterior epiphyseal line has produced a marked genu recurvatum, requiring a corrective osteotomy.

FIG. 4-D

Roentgenogram made when the patient was twenty-five years old, five years after corrective osteotomy.

TABLE I

CENTRAL-GRAFT FUSION OF TUBERCULOUS KNEES IN CHILDREN

Patient	Age (yrs.)	Sex	Duration of Disease	Fusion Time (mos.)	End Result	Angle of Fused Knee (degrees)	Limb Length	Follow-up Period (yrs.)
P. F.	7	M	2½ yrs.	5	Fused	5 flexion	½ in. short	9
P. R.	9	F	28 mos.	6	Fused	5 flexion	½ in. long	3½
B. B.	7	F	6 yrs.	6	Fused	4 flexion	½ in. short	6½
R. L.	11	M	1½ yrs.	4	Fused	8 extension	No shortening	9
S. D.	6	F	2 yrs.	5	Fused	4 flexion	½ in. long	5
P. G.	7	M	7 mos.	6	Fused	Straight	¾ in. long	10
C. H.	11	M	2¼ yrs.	6	Fused	5 flexion	⅞ in. short	5½
R. J.	10	M	2 yrs.	4	Fused	Straight	1¾ in. short	13
R. K.	9	M	1½ yrs.	6	Fused	30 extension—corrective osteotomy was refused	1½ in. short	15
L. R.	8	M	6 yrs.	5	Fused	5 flexion after two corrective osteotomies	1 in. short	8
L. F.	6	F	2 yrs.	6	Fused	Straight—after corrective osteotomy for 15° genu recurvatum	1⅞ in. short—procedure for epiphyseal arrest done on opposite leg	7
B. Z.	12	M	3¼ yrs.	6	Fused	5 flexion—after corrective osteotomy for 25° genu recurvatum	¾ in. short	10
P. S.	8	F	1¼ yrs.	6	Fused	10 flexion	½ in. short	8
J. R.	9	M	6 yrs.	5	Fused	Straight—after corrective osteotomy for 22° genu recurvatum	½ in. short	10
D. S.	13	F	6 yrs.	6	Fused	4 flexion	⅜ in. long	4

cartilage on the under side of the patella in children should be left untouched or, if the patella is diseased, total patellectomy should be done.

After the tourniquet has been released and careful hemostasis carried out, the wound is closed. Dry sterile dressings are then applied, followed by a toe-to-groin, plaster-of-Paris spica which is used for fixation.

The optimum position for the fused knee varies according to the patient's age and occupation. In children, it has been our custom to fuse the tuberculous knee in a straight position, but in adults we believe that 15 degrees of flexion should be the general rule, although this may vary more or less depending on the patient's occupation. In either case, the technique of operation is the same.

End Results of the Central-Graft Operation in Children

The statistical data concerning the fifteen children with central grafts of the knee are given in Table I. It will be noted that all of the fifteen tuberculous joints were successfully fused by this method. The limb length varied in the course of time after fusion—four limbs became a little longer, and eleven more or less shorter, as compared with the normal limb not operated on.

In the eleven limbs in which any shortening was evident, the amount was less than one inch in eight limbs and in the remaining three limbs it was one and three-quarters inches, one and one-half inches, and one and seven-eighths inches, respectively. Limb lengths were determined by both teleoroentgenogram and by direct measurement with a tape.

In trying to determine the cause of shortening, we have not been able to explain why some of the limbs that were operated on became slightly longer whereas others showed some shortening. It might be concluded that the inch or less of shortening that occurred in eleven patients was caused by: (1) destruction of bone resulting from the disease itself; (2) disuse and abnormal function of the leg associated with the knee fusion, which not only produced atrophy of the thigh

TABLE II

CENTRAL-GRAFT FUSION OF TUBERCULOUS KNEES IN ADULTS

Patient	Age	Sex	Duration of Disease	Fusion Time	End Result	Follow-up Period
M. D.	38	M	9 yrs.	4 mos.	Fused	8 mos.
M. C.	39	F	3 yrs.	6 mos.	Fused	10 yrs.
D. B.	22	F	2½ yrs.	5 mos.	Fused	15 yrs.
D. G.	52	F	9 yrs.	8 mos.	Fused	15 mos.
A. L.	50	M	4 yrs.	5 mos.	Fused	1 yr.
C. R.	22	M	1½ yrs.	5 mos.	Fused	12 yrs.
H. R.	30	F	2½ yrs.	5 mos.	Fused	5½ yrs
J. S.	23	M	10 mos.	(Non-union for 6½ mos.) fused 6 mos. after second operation	Fused	2 yrs. after second operation
F. W.	28	F	2 yrs.	6 mos.	Fused	8 yrs.

and hip muscles, but also decreased the circulation of blood, thus retarding the growth stimulus to the knee-joint epiphyses; or (3) varying degrees of retardation without actual arrest of growth, possibly caused by the operation and the presence of the central graft. We really do not know the exact answer, but can say that: (1) a bone graft passing through the central portion of an epiphysis, in our experience, has never caused cessation of bone growth, and (2) we have never seen a central bone graft cause premature closure of an epiphysis in any of the serial postoperative roentgenograms of these children.

Of the three patients in whom there was shortening of more than one inch, in two of them it was noted that there was marked genu recurvatum which was corrected by osteotomy in one patient; the other patient refused osteotomy. The shortening in these two patients can be accounted for on the basis of epiphyseal arrest of the anterior portions of the epiphyseal plates which had resulted from fusion of the patellae to the femoral condyles at the anterior epiphyseal lines (Fig. 4-A) in addition to the genu recurvatum. In the third patient (R.J.), the shortening could not be explained, since the last roentgenograms made six years after the fusion showed that both epiphyses at the fused knee were open and identical in appearance with those of the opposite normal knee joint. The epiphyses at the hip and ankle joints of this short limb also appeared normal.

Despite the shortening of one and three-quarters inches in this patient's limb, at the time of writing he has been working full time for years as a finishing polisher, and is very active. He is able to run and play ball and, according to him, has "no difficulty whatsoever." He denies any backache or hip pain and prefers no lift on his shoe because "it wears off too fast."

Complications

The most frequent complication associated with this operation in children was the occurrence of genu recurvatum, which developed in four cases. Three of these deformities were corrected by supracondylar osteotomies; the fourth patient with this deformity refused operation, saying that he was satisfied with his leg as it was.

There was one case of increase of flexion deformity after knee fusion (L.R.) which required two separate corrective osteotomies as the child grew older to obtain a final and permanent knee position of 5 degrees of flexion. We believe that the cause of this deformity was definite localized tuberculous disease at the posterior portion of the femoral epiphyseal line, producing epiphyseal arrest at this point.

In every one of our four patients in whom genu recurvatum developed, roentgenograms revealed that the undersurface of the patella fused to the femur across

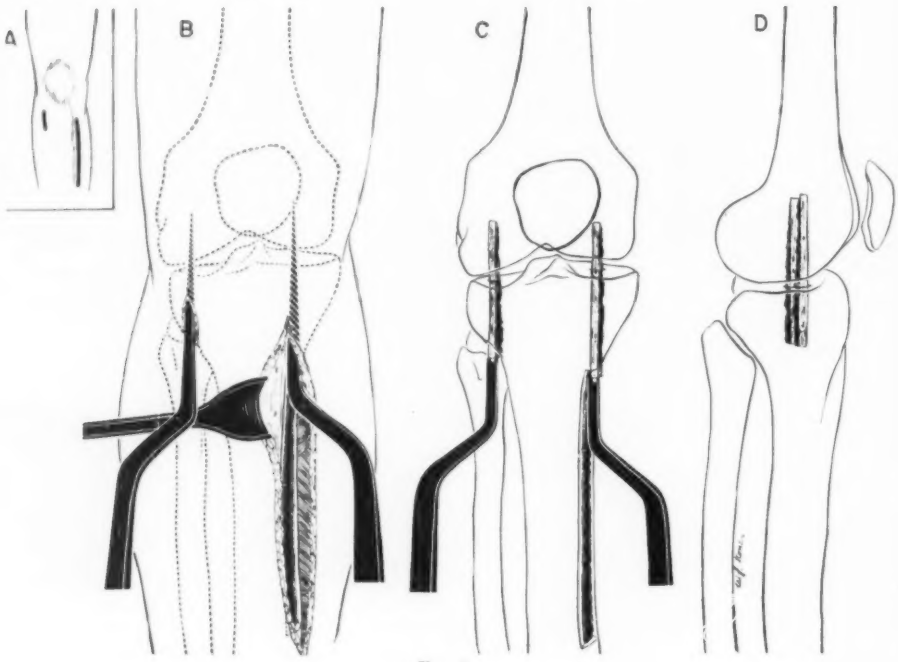

FIG. 5

Central-dual-graft fusion of the knee joint: *A*, skin incisions; *B*, bone tunnels made for bone grafts with the Hatt bayonet osteotomes; *C* and *D*, bone grafts being driven into place with Hatt bayonet drivers, shown in the anteroposterior and lateral views.

the lower femoral epiphyseal line as a bone graft, arresting growth anteriorly at that point.

After we made this discovery, we decided not to denude the undersurface of the patella in children when arthrodesis is performed, and as a result have not had any more instances of genu recurvatum.

End Results of Single-Central-Graft Operation in Adults

The results of the central-graft fusion of the knee in adults are shown in Table II. There were nine patients (four males and five females) with nine knee fusions; all of the operations were successful. One complication was encountered—a fracture of the central graft resulting in non-union and requiring a second operation, in which a central tibial graft was driven upward through a prepared tunnel across the articulating surfaces without opening up of the joint. This operation was successful and resulted in solid fusion six months later.

Dual-Central-Graft Operation for Tuberculous Knees in Adults

If the tuberculous involvement of the knee in an adult is of long duration with only a limited degree of painful motion (a jog) present in the joint, or if the patient is quite elderly and not considered able to withstand a long or open operation on the affected joint, we have employed a relatively simple procedure. This consists in driving two bone grafts parallel to each other and in the frontal plane across the articulation, thus locking it without incising the synovial lining or exposing the joint by extensive open operation. We have termed this procedure a dual-central-graft fusion and have performed it successfully on eight of ten patients.

TABLE III

CENTRAL-DUAL-GRAFT FUSION OF TUBERCULOUS KNEES IN ADULTS

Patient	Age	Sex	Duration of Disease	Fusion Time	End Result	Follow-up Period
A. M.	52	F	45 yrs.	6 mos.	Fused	6 yrs.
L. M.	46	M	9 mos.	(Non-union)	Failure	13 mos.
A. S.	18	F	1½ yrs.	4 mos.	Fused	5 yrs.
A. K.	37	F	4 yrs.	4 mos.	Fused	16 mos.
G. M.	58	M	2½ yrs.	3 mos.	Fused	7 mos.
J. H.	47	M	6 yrs.	4 mos.	Fused	9 mos.
A. M.	63	F	6 yrs.	(Non-union)	Failure	6 yrs.
K. M.	52	M	16 yrs.	(1 year although grafts broke at 4 mos.)	Fused	2 yrs.
J. D.	48	M	3 yrs.	4 mos.	Fused	18 mos.
K. B.	58	F	5 yrs.	5 mos.	Fused	1 yr.

The operation has naturally been restricted to adults, since in children it might produce epiphyseal arrest and interfere with growth.

The Technique of Dual-Central-Graft Operation

A vertical incision is made over the flat surface of the tibia beginning one inch below the tibial table and extending ten inches distalward (Fig. 5, *A*). This incision is deepened down to the bone, exposing the flat tibial surface, from which a bone graft is removed with a double-bladed electric circular saw. This graft should be eight inches in length and include the full thickness of the cortex. Its width should be that of the Hatt osteotome which will be used to make the trans-articular tunnel into which the graft is later driven.

After removal, the massive bone graft is cut into two grafts, four inches in length, which are preserved in saline solution or moist gauze while two bone tunnels are being prepared for them.

To prepare these tunnels, two guide wires, three-thirty-seconds of an inch, are driven upward across the joint at points where the upper tibial cortex flares out to form the inner and the outer tibial tables. The incision already made for removal of the bone graft will suffice for placement of the medial guide wire, but a second short incision parallel to the first and on the outer side of the leg is necessary to obtain adequate exposure for the placement of the lateral guide wire on the outer side (Fig. 5, *A*).

After these two wires have been inserted, a roentgenogram is made at the operating table to determine their direction and depth. If they lie in a vertical plane parallel to each other and penetrate the femoral condyles (about one inch) then and only then should their positions be considered satisfactory for the locations of the tunnels.

The tunnels are created by first cutting small slots in the cortex of the tibia at the sites where the guide wires enter the bone. These slots are cut with a small, fine osteotome so as to freely admit the sharp ends of the Hatt bayonet osteotomes which are driven upward along the course of the wires, across the joint and into the femoral condyles to the desired depth (Fig. 5, *B*).

Each graft is sharpened at one end and cut appropriately for length, after which their sharpened ends are directed into the tunnels and they are driven upward across the articular surfaces of the tibia and femur by means of a Hatt bayonet driver. In this way, the articulation is bridged and locked by two parallel bone grafts, without exposing the interior of the joint (Fig. 5, *C* and *D*).

If for any reason it should not be advisable to make any incisions in the upper end of the tibia, the same operative procedure can be carried out by approaching the joint from above through two small incisions and driving the bone grafts from above downward across the articulation. In such a case, the opposite

FIG. 6-A

Tuberculosis of the knee, of long duration and accompanied by pain on jogging.

tibia might have to be used as the graft donor site.

After closure of the wound, the leg is immobilized in a toe-to-groin, plaster-of-Paris single spica for four to six months or longer, depending on the rapidity of fusion as determined clinically and by roentgenograms.

End Results of Dual-Central-Graft Operation

Statistics regarding the patients treated with dual central grafts are recorded in Table III.

This operative procedure was employed in ten patients in whom there were eight successful fusions and two failures. In one patient (K.M.), whose bone grafts broke, there was delayed union. The cause of fracture of the grafts in this patient is not known, but was thought to be due to too early ambulation, which was started at four months after operation. As soon as the fracture was discovered, a plaster-of-Paris cylinder was applied to the extended limb and weight-bearing was permitted, which fortunately resulted in fusion one year after operation.

Of the two failures, one patient (L.M.) required two separate operations, and, at the end of two years, there were indications that fusion was going to occur when he died suddenly of a heart attack. At the time of the second attempted fusion, the disease in his knee joint showed increasing destruction of the tibial table and, although the grafts were firmly united to the femoral condyles, there was no sign of union with the tibia. In the other patient who is listed as a failure (A.M.), who was sixty-three years of age, there was definite non-union with absorption of the bone grafts, and it was not considered advisable to make another attempt at fusion, since her general condition was such that she would be bedridden anyway.

It is of some interest to point out that in two of the three patients with faulty fusion (A.M. with non-union and K.M. with delayed union) bank bone instead of autogenous tibial was used for the bridging grafts. In the remaining seven patients,

FIG. 6-B

One year after operation.

whose knees fused normally, autogenous grafts were employed (Figs. 6-A and 6-B).

TUBERCULOSIS OF THE ANKLE JOINT

Tuberculosis of the ankle joint responded quite well to fusion by means of the central-graft method. In no case was it necessary to do more than drive a tibial-bone graft across the central portion of the diseased joint or joints. The central position of the graft as it crossed any epiphysis did not cause closure of the epiphyseal plate or other abnormality as shown on the roentgenogram in any of these children. In no instance was the diseased ankle or any of the tarsal joints exposed for the removal of articular cartilage.

Central-Graft Operation for Tuberculous Ankles

The joint can be approached operatively either anteriorly or posteriorly, depending on the condition of the skin and whether or not the subtalar joint should be included in the fusion. If the latter joint is to be included, then the anterior approach is indicated (Fig. 7).

If, on the other hand, only the talus is to be fused to the tibia, either approach can be used, often depending on the skin condition. If, for instance, old healed sinus tracts or deep scars are present in front, then the bone graft should be inserted from behind, or *vice versa*.

By the use of a Kirschner guide wire and roentgenographic control, the bone tunnel can be easily and accurately positioned for reception of the intra-articular graft, and the operative wound required is only a small incision through the skin and soft parts down to the cortex of the bone, exposing an area large enough to admit a guide wire and the Hatt bayonet osteotome which creates the tunnel. A tibial graft the width of the constructing osteotome is then impacted in the tunnel opening (Figs. 7 and 8). It is, of course, a wise precaution to check by roentgenogram before closure of the wound to be certain that the graft has crossed the desired articular surfaces and is in its optimum position.

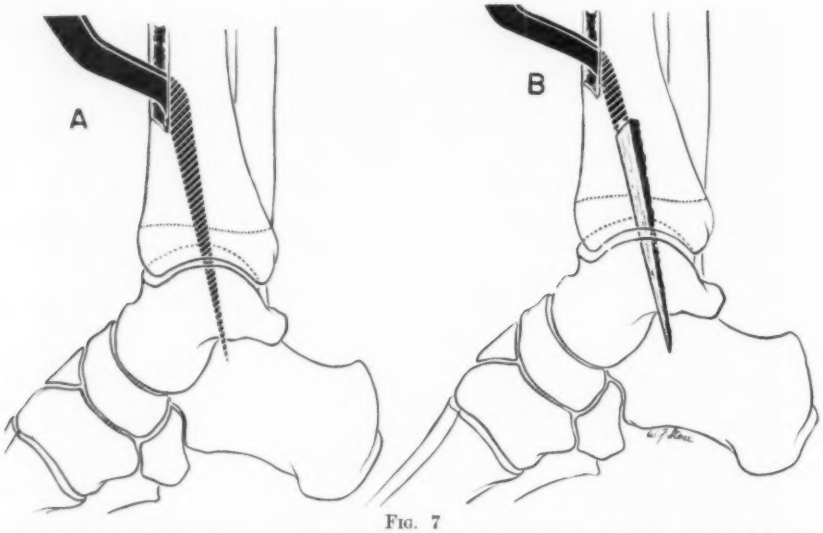

FIG. 7

Central-graft fusion of the ankle and subtalar joints: *A*, creation of the bone tunnel for the central bone graft; *B*, graft being driven into place.

End Results of the Central-Graft Operation in Tuberculous Ankles

Nine patients with tuberculosis of the ankle and tarsal bones were treated by the central-graft operation (Table IV). Four patients were children and five were adults, and in all the disease had been present over a period of years before they were operated on. This long duration of the disease may explain why no erasion of joint cartilage was necessary for fusion to be obtained. In all probability, the disease itself had destroyed sufficient joint cartilage to permit fusion with the aid of the bridging bone graft. In any case, fusion was successful in all

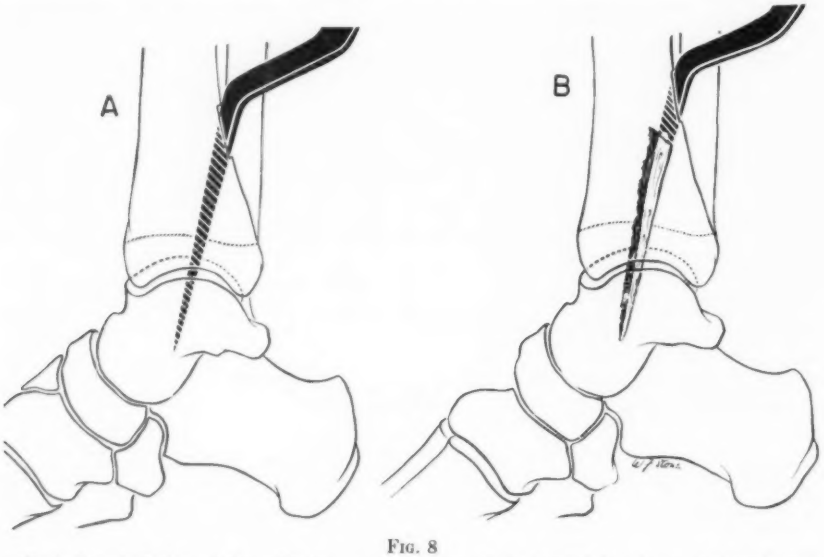

FIG. 8

Central-graft fusion of the ankle joint: *A*, creation of the tunnel for the central graft with the Hatt bayonet osteotome, by the posterior approach; *B*, graft being driven into place.

TABLE IV

CENTRAL-GRAFT FUSION OF TUBERCULOUS ANKLES

Patient	Age	Sex	Duration of Disease	Fusion Time	End Result	Follow-up Period
E. N.	18	M	2½ yrs.	4 mos.	Fused	9 yrs.
A. G.	45	F	25 yrs.	6 mos.	Fused	1 yr.
G. F.	71	M	1 yr.	6 mos.	Fused	3 yrs.
J. V.	35	M	2 yrs.	6 mos.	Fused	3 yrs.
R. P.	5	M	2 yrs.	10 mos.	Fused	4 yrs.
R. W.	7	M	3 yrs.	4 mos.	Fused	10 yrs.
A. L.	13	M	7 yrs.	4½ mos.	Fused	6 mos.
E. H.	53	F	10 mos.	6 mos.	Fused	1½ yrs.
D. G.	5½	F	5 yrs.	6 mos.	Fused	10 yrs.

nine patients by means of this rather simple and non-traumatic operative procedure of central-graft fusion.

Representative examples are shown in Figures 9-A, 9-B, 9-C, 10-A, and 10-B.

TUBERCULOSIS OF THE ELBOW

Central bone-grafting was not so successful in tuberculosis of the elbow as it was in the knee or ankle. Only six patients were treated by this method; the results are recorded in Table V.

FIG. 9-A

Roentgenogram made on admission of patient, showing acute active tuberculosis of the ankle with draining sinuses.

TABLE V

CENTRAL-GRAFT FUSION OF TUBERCULOUS ELBOWS

Patient	Age	Sex	Duration of Disease	Fusion Time	End Result	Follow-up Period
C. B.	50	M	10 yrs.	(Non-union for 1 yr.) fused in 3 mos. after second operation	Fused	1 yr. after second operation
M. C.	53	F	3 yrs.	4 mos.	Fused	7 yrs.
N. K.	21	M	3 yrs.	6 mos.	Fused	16 yrs.
A. M.	28	F	2 yrs.	6 mos.	Fused	9 yrs.
M. Z.	63	F	1 yr.	(Non-union; patient refused reoperation)	Non-union	1 yr.
W. M.	15	M	10 yrs.	(Non-union, 13 mos.) fused 6 mos. after second operation	Fused	1½ yrs.

Central-Graft Operation for Tuberculous Elbows

The technique of inserting the central graft in the elbow is to approach the joint from behind through a small incision over the mid-portion of the olecranon. A Kirschner wire, three-thirty-seconds of an inch, is drilled through the olecranon, across the joint to pass into the medullary canal of the distal shaft of the humerus (Fig. 11). Roentgenograms are then made (in the operating room) to make sure that the wire is in proper position to serve as a guide for the construction of the tunnel for the graft.

As soon as the position of the guide wire is seen to be suitable, a small window is cut in the cortex of the olecranon with a fine osteotome, at the point where the wire penetrated the surface of the bone. The size of the window is made to freely admit the tip of the Hatt bayonet osteotome that is to be used in creating

FIG. 9-B

Roentgenogram made five years later, showing marked improvement with healing of all sinuses, thus permitting operation for fusion.

FIG. 9-C

Roentgenogram of the ankle one year after the operation for fusion.

the tunnel. By following the course of the guide wire, the tunneling osteotome is driven through the olecranon, across the diseased joint surfaces, and into the substance of the humerus (Figs. 11, 12-A, and 12-B).

Repeat roentgenograms are then made to ascertain that the alignment and depth of the tunnel are satisfactory for the insertion of the central bone graft,

FIG. 10-A

FIG. 10-B

Fig. 10-A: Roentgenogram showing tuberculosis of the ankle and subtalar joints. The Hatt osteotome is shown in the preparation of a tunnel for the central bone graft.

Fig. 10-B: Seven years after the operation for central-graft fusion of the ankle and subtalar joints.

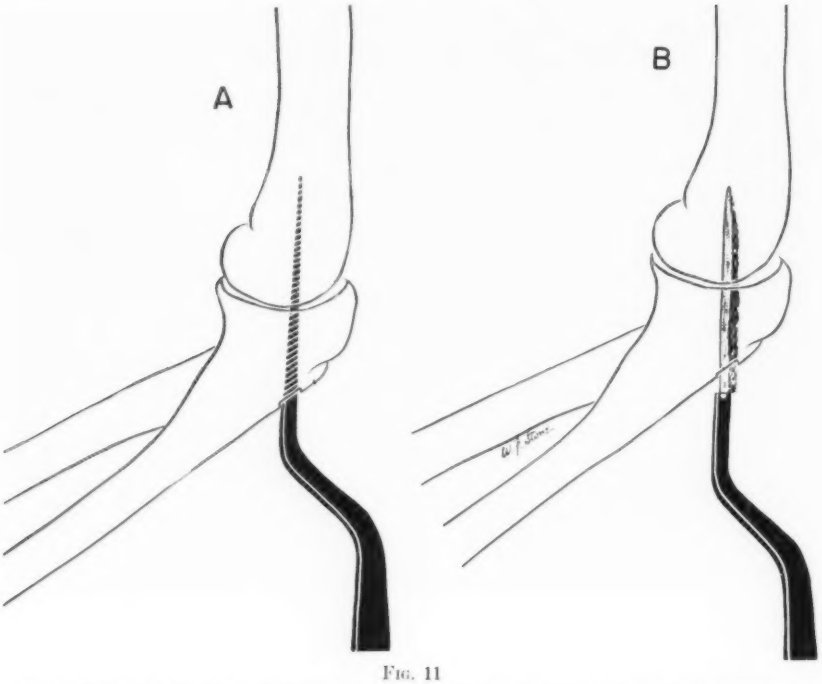

FIG. 11

Central-graft fusion of the elbow joint: *A*, creation of the tunnel for the central graft with the Hatt bayonet osteotome; *B*, driving the graft into position.

which is usually obtained from the patient's tibia or fibula. In this series of cases, the upper end of the radius with its articulation was left untouched, since it did not appear to be extensively involved in the tuberculous process, and the disease was quiescent.

After closure of the wound, the arm is placed in a plaster-of-Paris shoulder spica for external immobilization. After three or four months the shoulder spica is replaced by a long arm cast until solid bone union is revealed by roentgenograms.

End Results of the Central-Graft Operation in Elbows

In three patients there was failure to get union after the initial operation. One of these patients (C.B.) when he was first seen by us had a flail elbow with marked loss of bone substance and draining sinuses as the result of an earlier operation for excision of the elbow joint for tuberculosis. The joint was so painful and useless that the patient himself strongly urged an attempt at fusion even though he was told that the chances of success seemed very doubtful. After several months of conservative treatment, including incision and drainage, antituberculosis drugs, and plaster-cast immobilization, the sinuses healed and the disease appeared to become quiescent. At this time a central-graft fusion was performed, but the bone graft fractured and, after a period of eleven months, it was decided that union would not take place. It also became apparent that in order to obtain a successful fusion, a degree of immobilization greater than that from a plaster cast would be necessary and, therefore, at the second operation a stainless-steel plate was shaped and used for internal fixation with screws, in addition to a new central-bone graft. The added fixation apparently solved the problem, and fusion proceeded quite rapidly, but the successful final result could not be credited to

FIG. 12-A

Roentgenogram showing tuberculosis of the elbow before the central-graft operation for fusion.

central-graft fusion alone, and the case was therefore listed as a failure.

The second failure occurred in a man, sixty-three years old (M.Z.), who had been suffering from tuberculosis of the elbow for a year. The roentgenographic appearance of the diseased joint four months after operation was disappointing, and reoperation was seriously considered and advised. The patient, however, refused a second operative procedure, saying that he was content with the joint as it was, with a limited degree of painless motion (a jog).

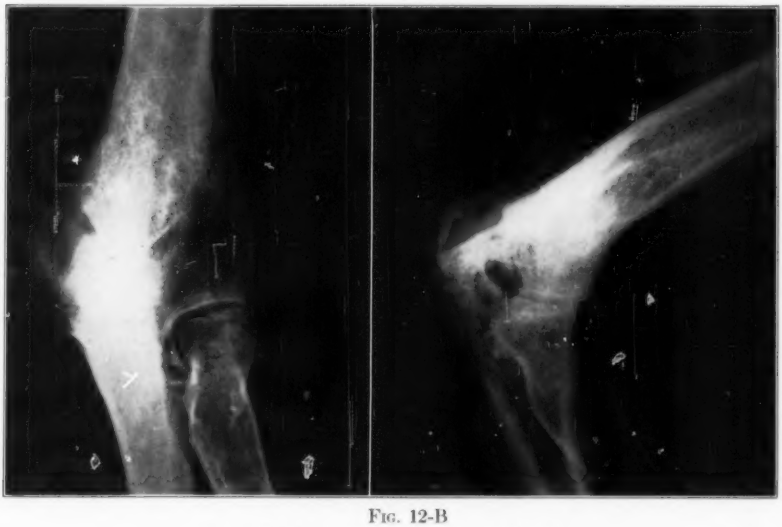

FIG. 12-B

Twelve years after the operation.

The third failure (W.M.) was due to a fracture of the bone graft five months after operation, but a second operation, similar in character, resulted in bone fusion.

Thus, of the six elbows that were treated by the central-bone-graft type of fusion, five eventually fused, but two of these five required a second operation, and the sixth elbow remained with non-union which one year after operation was asymptomatic with a limited degree of painless motion (a jog).

The majority of these patients with tuberculosis of the elbow were treated before the advent of antituberculosis drugs. Nowadays, with the help of these drugs, conservative treatment is often successful, resulting in a joint with adequate motion and useful function. Two of the patients, however, whose elbows were recently fused, had previously been treated by resection of the joints for tuberculosis before they came to us. The results of this procedure were so unsatisfactory that these patients begged for a firm stiff joint, which was successfully obtained in both instances.

The experience of one of us (G.V.G.) with excision of the elbow joint for tuberculosis has not been a happy one, and the opportunity to see any good results from this operation in tuberculous elbows has not occurred.

At the present time, with the use of antituberculosis drugs, it is hoped that surgery may no longer be necessary in the treatment of tuberculosis of the elbow.

SUMMARY

The central-graft fusion operation, originally described by Hatt, in our experience has proved its value and has been most successful in the treatment of tuberculous knees and ankles, and reasonably successful in the treatment of tuberculous elbows.

In children, especially, the operation is of supreme value because it offers a high percentage of successful joint fusions without causing cessation of bone growth. In the series of cases just reviewed, solid bone fusion by this operation was obtained in all of the fifteen children with tuberculosis of the knee.

In the fusion of tuberculous knee joints in children it is of extreme importance to avoid allowing the patella to become attached to the femur at the anterior epiphyseal line; in our series of cases, this produced local cessation of growth with resulting marked genu recurvatum and shortening.

In adults, the single-central-graft fusion operation for tuberculosis of the knee was successful in all of the nine patients operated on, although one patient required a second operation. In the ten adults who underwent the more conservative dual-central-graft operation for tuberculous knees, successful fusions occurred in eight patients and failure occurred in two.

In the treatment of tuberculosis of the ankle, the central-graft fusion operation was performed in four children and five adults. Fusion was obtained in all of the patients, and we have had no failures. Also, it is of interest to know that this success was obtained without opening up of the diseased joints or erasion of the joint cartilages.

In tuberculosis of the elbow, the operation was not quite so successful as it has proved to be in the cases of disease of the knee and ankle. Six patients with tuberculosis of the elbow were operated on, and although fusion was obtained in five of them with the use of the central graft, a second operation was required in two of the patients. The one failure remained a non-union, and the patient declined another operative procedure because he was content with the result that he had.

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Absence of Motion at the Fourth and Fifth Lumbar Interspaces in Patients with and without Low-Back Pain*

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The subject of spinal mobility is first recorded in the work of Weber and Weber in 1836. Since that time, many investigators have presented their work in this field. Objectives, methods, and conclusions have varied considerably.

Our purposes are to attempt to evaluate the significance of absence of motion at the fourth and fifth lumbar interspaces found in 527 consecutive patients with low-back pain seen in private-office practice and to compare the findings with a control group of ninety-four healthy individuals.

As far as we can ascertain, a normal range of motion has not been established adequately for the fourth and fifth lumbar interspace levels, and we have made no attempt to determine the normal range of motion. Tanz, in 1953, found a considerable variation in the amount of motion at these levels similar to that noted in both our control and low-back pain series. Therefore, it is our present feeling that quantitative changes from subject to subject have no significance. Consequently, we have used either presence or absence of motion as the basis of our investigation.

Our plan of study, after history and clinical examination, included roentgenographic examination by a standard procedure. Besides the routine anteroposterior, lateral, stereoscopic obliques, spot-lateral lumbosacral, and "up-shot" view in the lumbosacral plane, lateral flexion and extension roentgenograms were made with the patient in the upright, sitting position. This method is similar to that first described by Ferguson, and recently used by Morgan and King, Hasner and associates, Allbrook, and Jirout.

TECHNIQUE FOR BENDING STUDIES

The patient is seated on a stool in front of an upright Bucky apparatus in proper position for a lateral roentgenogram of the spine. His feet are placed comfortably on a small footstool so that flexion and extension of the spine can be obtained without moving the pelvis or lower extremities. Further immobilization can be obtained by using a firm, radiolucent sling which passes over the thighs and holds the patient firmly against the stool, the sling passing below the iliac crests. The tube is centered at the level of the third intervertebral-disc space and film exposures are made with the trunk in maximum flexion and extension.

The presence or absence of motion is ascertained from the roentgenograms by superimposing the sacrums in the two films and determining the difference of the angles formed at the lumbosacral junction. The fifth lumbar bodies are then superimposed and motion between the fifth and the fourth vertebrae is determined in the same fashion. In the absence of motion all bodies are superimposed.

As the method devised by Morgan and King of making flexion and extension studies for primary instability is practically the same as that which we have described, we recorded the presence or absence of so-called primary instability. Morgan and King defined primary instability as anteroposterior sliding of one vertebra on another in the absence of any other roentgenographic abnormality of the discs or vertebrae. Their method of measurement is quoted as follows:

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, New York, N. Y., February 5, 1958. Part of this material was presented at the Western Orthopedic Association, October 1956, and as Trinity Lecture, Oxford University, June 1957.

"The instability can also be determined by drawing a line along the front border of each individual vertebral body. Instability exists if the line does not pass close to the anterior lip of the vertebral body (epiphysial bone ring) immediately below and above it. . . . This test is accurate for most people in whom flexion and extension take place mainly at the lumbo-sacral junction."[10]

ANALYSIS OF SERIES

Our 527 cases (393 men and 134 women) were separated into the following clinical classifications for the purpose of analysis:

A. Sprains: Patients with pain after trauma who show no roentgenographic evidence of bone injury or pathological change, but who have clinical findings of low-back disability.

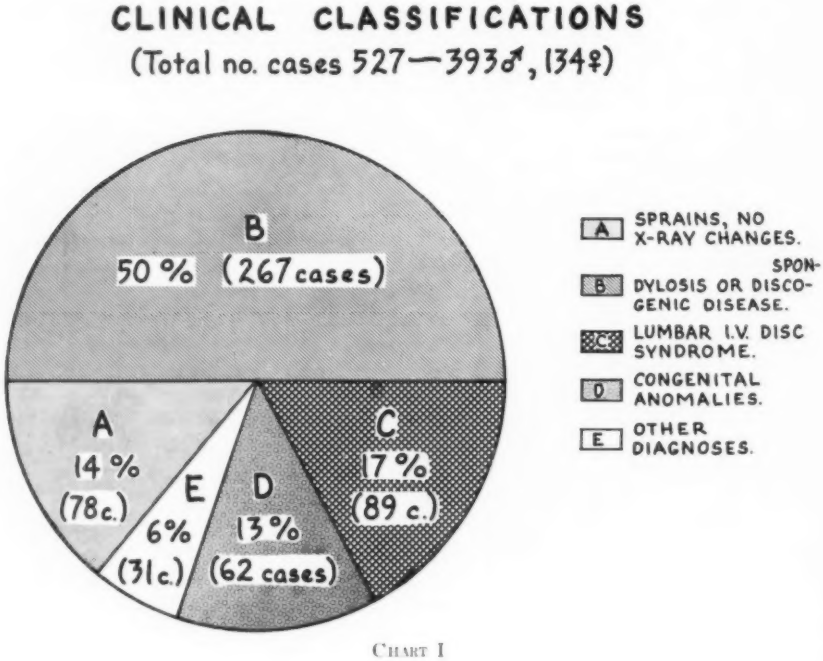

CHART I

B. Spondylosis, disc degeneration, or both: Patients with pain after injury who have clinical findings of low-back disability with no roentgenographic evidence of bone injury but with roentgenographic manifestations of either or both of these related chronic, degenerative changes.

C. Lumbar intervertebral-disc syndrome: Patients with typical back and leg pain who show sufficient positive clinical evidence of nerve-root pressure to warrant the clinical diagnosis of disc protrusion without confirmatory myelogram. Consideration was given to the type, onset, and localization of the low-back pain, tenderness, limitation of spinal motion (especially the flattening of the lumbar arc on forward bending), straight-leg raising, reflex changes, sensory changes, motor weakness, and atrophy.

D. Congenital anomalies: Patients with low-back pain without roentgenographic evidence of bone injury but with congenital or developmental anomalies restricted to the lower two lumbar vertebrae and sacrum. These anomalies were limited to spondyloschisis—unilateral or bilateral—with or without spondylolisthe-

sis, and transitional lumbar or sacral vertebrae. Asymmetry of facets, spina bifida, and other similar anomalies were not included.

E. Other diagnoses:

1. Old, healed lumbar-body fractures not limited to the fourth and fifth lumbar vertebrae.

2. Objective postural defects without roentgenographic or clinical evidence of injury or disease, including increased lumbar lordosis, forward or backward pelvic inclination, with or without relaxed abdominal musculature, and inequality of leg length. Patients are placed in this category by elimination of all other possible factors.

3. Psychosomatic, that is, patients without clinical or roentgenographic evidence of orthopaedic disability and with stigmata of functional overlay.

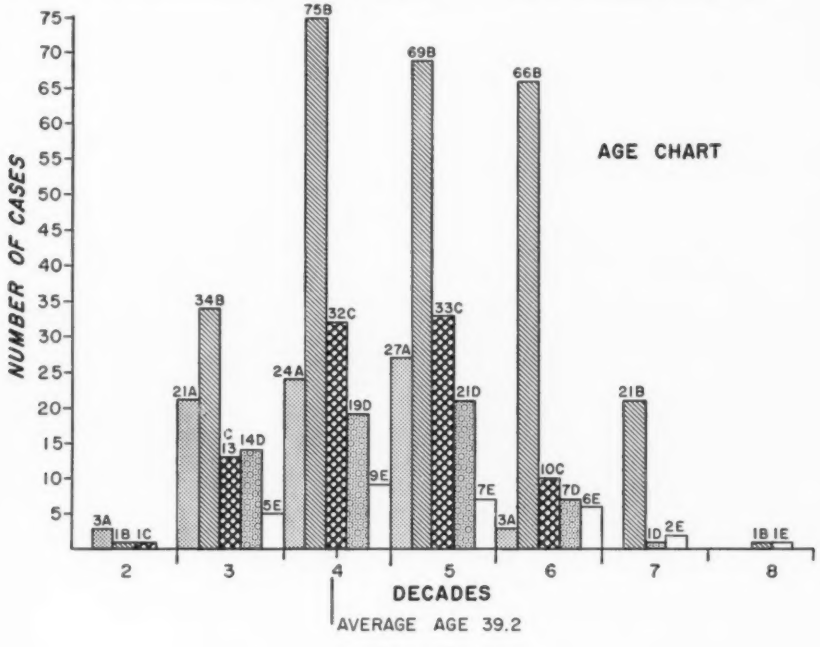

CHART II

For key, see Chart I.

4. Scoliosis, that is, patients with clinical and roentgenographic evidence of abnormal curvature. This includes paralytic scoliosis, congenital scoliosis, and idiopathic scoliosis.

5. Other vertebral bone diseases including osteoporosis and Paget's disease (one case). No neoplasms were discovered in this series (Chart I).

The over-all average age was 39.2 years, the youngest being seventeen, and the oldest, eighty-two. Chart II tabulates by decade the number of cases in each classification. Two-hundred and forty-nine patients (47 per cent) complained of pain referred down one or the other lower extremity. Further analysis disclosed that according to our criteria in 68 per cent of the patients with this complaint symptoms could not be attributed to the lumbar intervertebral-disc syndrome.

Flexion and extension roentgenograms of the 527 patients showed mobility at both levels in 44 per cent and no motion in 43 per cent. There was primary instability in 13 per cent of 504 patients.

Analyzed by the same methods, the ninety-four normal controls showed mo-

bility at both levels in 77 per cent, no motion in 15 per cent, and primary instability in 8 per cent (Chart III).

DURATION OF SYMPTOMS

Symptoms of more than two months' duration were classified arbitrarily as chronic, whereas those of less than two months' duration were considered acute. On this basis 36 per cent of the cases were acute, and 64 per cent were chronic. The duration of symptoms in the various diagnostic categories is given in Chart IV.

ROENTGENOGRAPHIC EVIDENCE OF LIMITATION OF LUMBAR SPINAL MOTION

Analysis of the patients with absence of motion by roentgenogram shows a consistent pattern, which is related to the duration of the complaint and not to

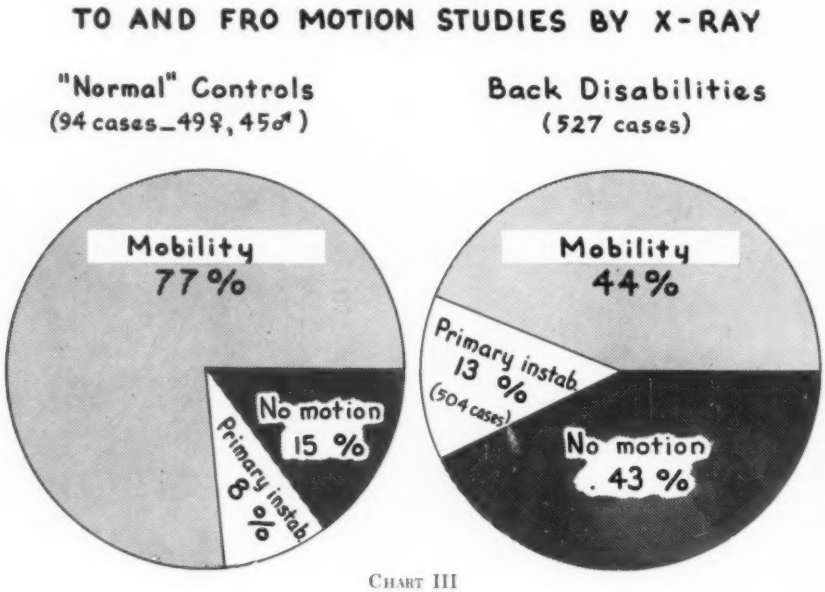

CHART III

the etiological classification. Absence of motion at one or both of the two lower lumbar vertebral levels was demonstrated in 226 of the total series of 527 patients. In these 226 patients, absence of motion occurred more frequently in patients with symptoms of over two months' duration, and in patients in the fourth, fifth, and sixth decades of life (Chart V).

RELATIONSHIP OF CLINICAL TO ROENTGENOGRAPHIC EVIDENCE OF LIMITATION OF MOTION

Since the spine is able to compensate for restriction of motion in one area, it does not follow that loss of motion, roentgenographically, will be accompanied by a measurable loss of motion in the back on physical examination. Of course, part of this discrepancy may be due to our inability to perceive minor changes in motion by physical examination.

There was no constant relationship between clinical and roentgenographic evidence of loss of mobility. In fact, among the 226 patients with roentgenographic evidence of loss of motion at one or both of the fourth and fifth lumbar interspaces, there was a relatively small percentage who showed clinical evidence of limitation of motion (Chart VI).

DISCUSSION

From a review of the available literature, it seems evident that the majority of the authorities concluded that the lower lumbar vertebral segments show roentgenographic evidence of restriction of motion after disease, injury, or both. Hibbs and Swift, in 1929, reawakened interest in the importance of abnormal biomechanics as a cause of low-back pain, stressing developmental abnormalities. Ferguson, in 1934, studied lumbosacral anomalies and emphasized the lack of potential reserve in these structures when subjected to abnormal stress. DeForest Smith, in the same year, suggested that a relationship exists between so-called reverse spondylolisthesis and hypermobility due to anatomical asymmetry of the articular

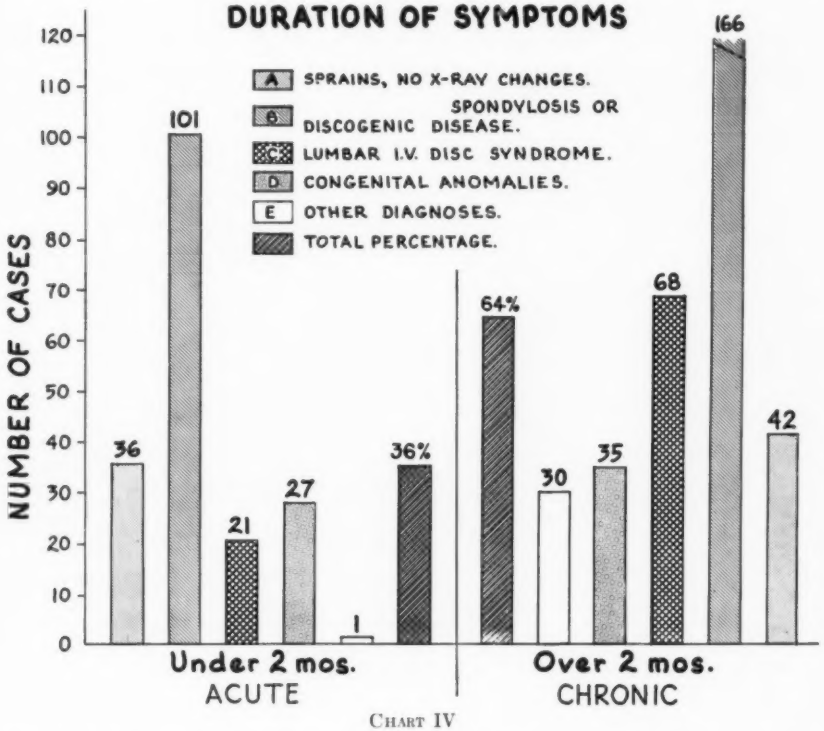

CHART IV

facets. Both Ferguson and DeForest Smith used roentgenographic motion studies to support their conclusions. With this stimulation of interest in this problem, Willis, in 1935, claimed reverse spondylolisthesis to be an optical illusion. It is interesting that tweny-three years later Melamed still stressed the uncertainties that arise in the roentgenographic diagnosis of true retrodisplacement at the lumbosacral junction.

In 1942, Duncan and Hoen demonstrated lack of spinal mobility at the level of the involved herniated disc. Knutsson, in 1944, could not verify Duncan and Hoen's findings, although it is not made entirely clear whether or not he was studying patients with disc protrusion or with disc degeneration. Begg and Falconer, in 1949, supported Duncan and Hoen's initial studies. Duncan and Hoen attributed limitation of motion at the site of an involved intervertebral disc to paravertebral muscle splinting. They used roentgenograms made in the upright position with the trunk in flexion and extension after the method described in 1934 by Ferguson.

In 1950, Tanz studied fifty-seven random patients (a high percentage having low-back complaints) and concluded that lumbosacral motion decreased with age. He found that 30 per cent of his patients over thirty-five years of age showed absence of motion at the lumbosacral junction. In 1953, he attempted to obtain a normal pattern of motion by studying ten children and forty-five adults, but he found a wide individual variation. Approximately 20 per cent of the normal adults showed absence of motion at the lumbosacral junction.

Jirout, in 1957, studied spinal motion in 100 normal individuals between fifteen and twenty-five years of age, also using roentgenograms made in the upright position with the trunk in flexion and extension. He considered 89 per cent to have normal dynamics, whereas 11 per cent showed disturbances of spinal mobility.

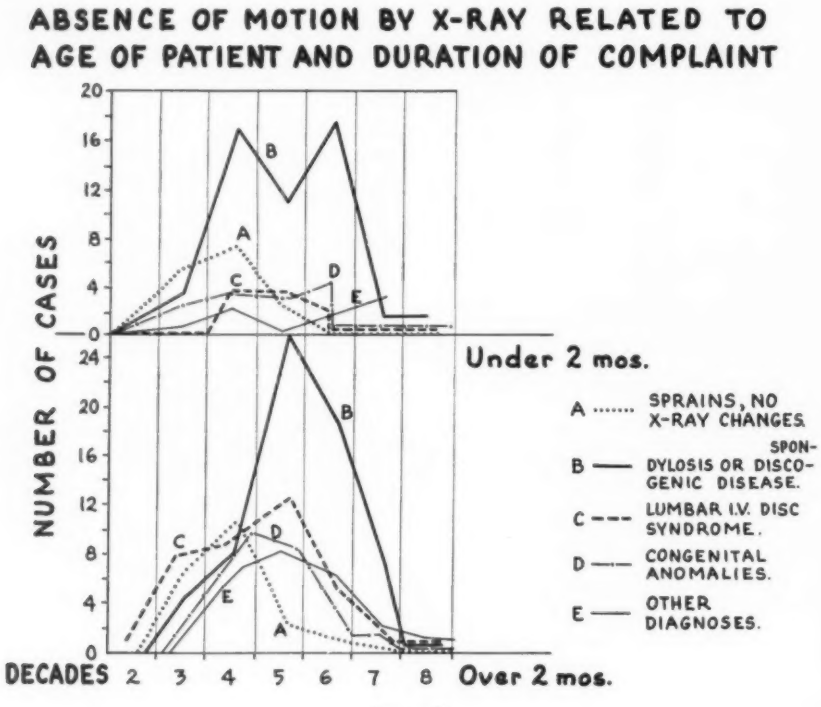
ABSENCE OF MOTION BY X-RAY RELATED TO AGE OF PATIENT AND DURATION OF COMPLAINT
NUMBER OF CASES
DECADES
Under 2 mos.
Over 2 mos.
A ..... SPRAINS, NO X-RAY CHANGES.
B —— SPONDYLOSIS OR DISCOGENIC DISEASE.
C --- LUMBAR I.V. DISC SYNDROME.
D —·— CONGENITAL ANOMALIES.
E —— OTHER DIAGNOSES.

CHART V

Allbrook, in 1957, using similar roentgenographic methods in thirty-two normal individuals, concluded that the fourth and fifth lumbar vertebrae undergo the greatest movement during flexion and extension, and that the range of motion decreases in the cephalad direction. This conclusion is contrary to previous investigations and to our own findings.

Hasner and associates, in 1952, stressed the evaluation of motion studies in the upright position as an important method for determining abnormal reactions in the ligamentous, articular, and muscular systems (the supporting apparatus of the spine) which are responsible for the majority of symptoms. In an effort to determine the role of muscle spasm in immobilizing the lower lumbar segments, they repeated their roentgenographic studies under general anesthesia and curarization in five patients and found that immobility remained. In an unspecified number of patients, an attempt was made to reduce muscle spasm with morphine and scopolamine sedation. This medication caused an insignificant improvement

of function. They concluded that "the myogenic inhibition of mobility is also associated with definite structural fixation."

Morgan and King, in 1957, reviewed low-back pain in 500 consecutive cases by means of flexion and extension roentgenograms made with the patients in the upright position. They found abnormal motion or primary instability at the lower two lumbar interspaces (which they termed primary instability) in 29 per cent of their series. Five hundred and four cases in our series were studied by their method. In 13 per cent of the patients with back pain and 8 per cent of the normal controls there was so-called primary instability.

We believe that there are sufficient observations now available to state that a small (Jirout, 11 per cent; Tanz, 20 per cent; our series, 15 per cent), but definite, percentage of individuals without back complaints have restriction of spinal

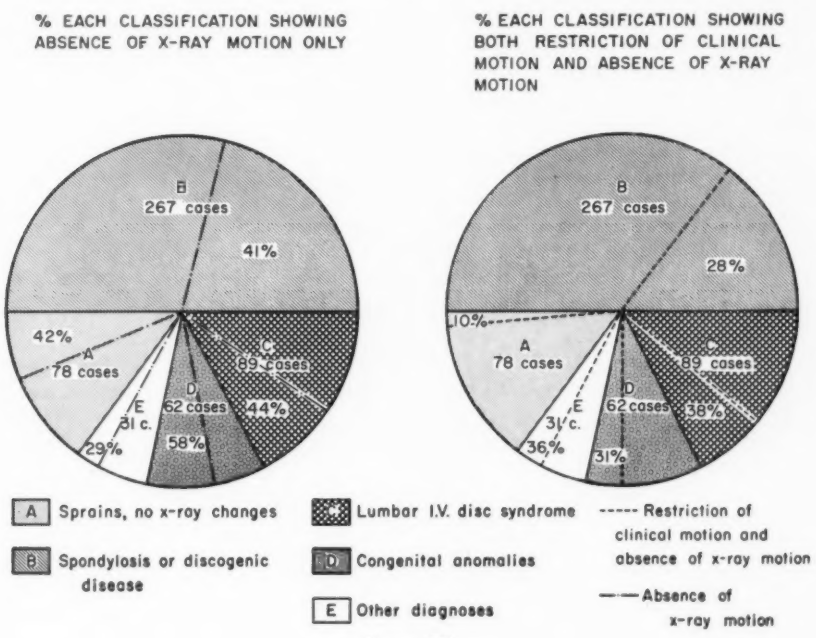

CHART VI

mobility at the fourth and fifth lumbar levels. Jirout poses the question whether these abnormal spinal dynamics in healthy individuals may indicate a predisposition to intervertebral disturbances. One of his patients who was normal in 1952 was re-examined in 1955 because of low-back pain and was found to have absence of motion at the fourth lumbar interspace.

In patients with back complaints and loss of spinal mobility on roentgenographic examination, it has not been established whether this restriction of motion is caused by pathological changes of bone, muscle spasm, or both. The work of Hasner seems to suggest that muscle spasm is not an important factor. It is our impression that architectural changes in bone, with the exception of certain congenital anomalies, rarely limit spinal mobility. In our series approximately 50 per cent of the patients with degenerative changes, that is, those in Classification *B* disclosed an excellent range of spinal mobility. No attempt was made to correlate the range of motion with the degree and type of degeneration.

In the light of this analysis it would appear that there is a direct relationship between loss of spinal mobility in the lower lumbar segments, the duration

of the back symptoms, and the age of the patient. In patients under thirty with strains (Classification *A*) loss of motion is practically absent in those with symptoms of less than two months' duration. In those with chronic symptoms (of more than two months' duration) loss of motion occurs at an earlier age and with more frequency (Chart V). A less definite but similar relationship to age and chronicity of symptoms is apparent in the patients in the other etiological classification.

The cause and significance of this loss of mobility are yet to be determined. Presumably it is the result of trauma and the natural wear and tear of daily activity. If it is not due to degenerative changes and muscle spasm, it may be due to contractures of the peri-osseous tissues. Should this prove to be the case, this might be one explanation for the observed beneficial effects of manipulative therapy since manipulation might be expected to correct these contractures.

CONCLUSIONS

1. Absence of motion at the fourth and fifth lumbar vertebral levels as seen in flexion and extension roentgenograms was found in 15 per cent of normal patients and in 43 per cent of patients suffering from low-back pain.

2. Abnormal mobility, so-called primary instability, was observed in 13 per cent of the patients with back symptoms and in 8 per cent of the normal controls.

3. The observed loss of spinal mobility seems to be related to the age of the patient and to the duration of the symptoms.

4. There is no definite evidence available as to the cause of this loss of mobility, but it is the authors' impression that changes in the peri-osseous soft tissues are responsible, rather than muscle spasm or degenerative changes.

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Epiphyseal Fractures

A Microscopic Study of the Healing Process in Rats *

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Although epiphyseal fractures are of common occurrence, microscopic studies of the healing process of these lesions have been few. This is in striking contrast to the vast numbers of reports describing the healing process of fractures through bone. Of the clinical studies of epiphyseal injuries, those of Aitken are of particular interest. His classification of these fractures is based on the course taken by the fracture line through the epiphyseal plate. Of the three varieties of epiphyseal fractures described by Aitken, the Type I injury is the most common (Fig. 1). This

Fig. 1

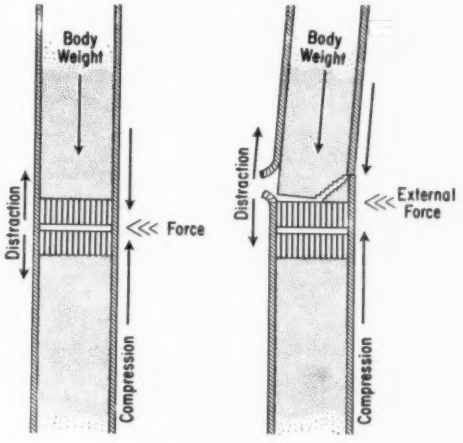

Fig. 2

Fig. 1: Type I fracture. The fracture line enters the hypertrophic zone of the cartilage plate, involves the deeper layers near the apex, at the center of the plate, and emerges through the metaphyseal trabeculae on the compression side. (Reproduced with permission from "Epiphyseal Fractures of the Lower Extremity." By H. R. Brashear, Jr., Southern Med. J., **51**: 845-851, 1958.

Fig. 2: Diagram of forces involved in Type I fracture.

lesion is usually caused by an angulating force. The fracture line travels horizontally within the plane of the epiphyseal plate, usually for most of its width, then leaves the cartilage to include a triangular segment of metaphyseal bone. The present study is limited to this Type I fracture.

The epiphyseal cartilage rests on a plate of bone in the epiphysis, here referred to as the subepiphyseal cartilage bone plate. Its principal function is apparently to support and protect the growth cartilage [2]. Four zones of cells may be identified in the epiphyseal cartilage. In the first, or resting, zone, which lies directly adjacent to the bone plate, the cells are irregularly placed. In the second, or zone of cell columns, orderly linear arrangement of the cartilage cells occurs. Only in the

* Read at the Annual Meeting of The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 27, 1959.

FIG. 3-A

FIG. 3-B

Fig. 3-A: Roentgenogram of the fracture produced at the distal femoral epiphysis of a rat.

Fig. 3-B: Roentgenogram of the same femur eight days after fracture, showing the widened distal epiphyseal plate.

resting zone and in the first part of the zone of cell columns have mitotic figures been noted [6]. In the third, or hypertrophic cell zone, the cartilage cells enlarge at the expense of their extracellular support. Here, as shown by Haas and later by Harris, is the weakest portion of the epiphyseal plate. This is a weakness, however, to bending, shearing, and tension stresses and not to compression forces. The fourth layer of cells, where the appearance of calcium salts in the matrix adds strength, is termed the zone of provisional calcification. This zone is invaded by capillaries, and the calcified spicules of cartilage matrix are included in the fine bone trabeculae that characterize the process of enchondral ossification.

By incising the periosteum about the epiphyseal plate, Haas demonstrated that the epiphysis could easily be separated from the metaphysis and that the line of separation was consistently within the zone of hypertrophic cartilage cells. It is to be noted, however, that the forces involved in this experiment were only angulation and distraction. This is not the case in the clinical Type I injury, which occurs typically in the youthful high-school football player who is struck on the lateral side of the knee. The blow tends to angulate the knee into a valgus position, resulting in a distracting force on the medial side of the epiphyseal plate. The medial portion of the fracture line thus lies, as would be expected, within the hypertrophic zone. However, the body weight and the intact medial periosteum, acting as the fulcrum of a lever, produce a compression force on the lateral side of the epiphyseal line (Fig. 2). The more elastic cartilage cells withstand the compression better than the small metaphyseal bone trabeculae, and the fracture line leaves the epiphyseal plate to include the lateral triangle of metaphyseal bone. The size of this triangular fragment seems to depend on the magnitude of the compression force. The greater the longitudinal thrust of body weight, and the stronger the periosteum, the larger the metaphyseal fragment of bone. On the other hand, if the periosteum ruptures easily and the longitudinal thrust is mild, the compression force is lessened and the lateral metaphyseal bone fragment is small.

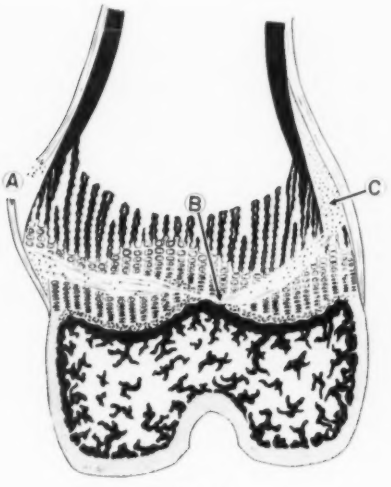

FIG. 4

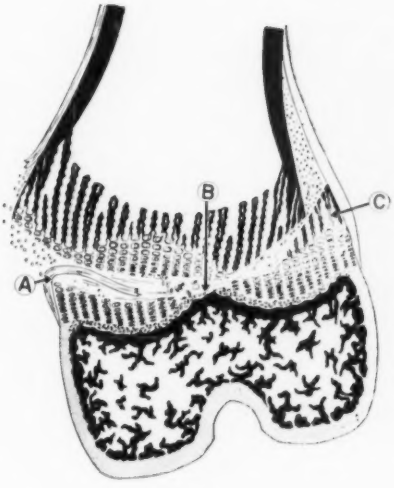

FIG. 5

Fig. 4: *A*, rupture of periosteum on distraction side; *B*, involvement of deeper layers of cartilage cells near the apex; and *C*, elevated periosteum on compression side with subperiosteal hemorrhage.

Fig. 5: *A*, periosteum trapped within the fracture; *B*, resting zone of cartilage cells frequently involved in a fracture near the apex; and *C*, triangular fragment of bone on the compression side.

Another force must be considered. Once the medial periosteum has ruptured, the external force acting on the leg and the longitudinal thrust of body weight may combine compression and shearing to result in a grinding of prominent portions of the epiphyseal plate, injuring any or all of its various zones. This might be suspected in the more markedly displaced Type I injuries in which the metaphyseal fragment of bone is small.

METHOD OF CREATING FRACTURES

Fractures of the distal femoral epiphysis were created in albino rats. Forty-two animals weighing between 100 and 165 grams were used. The rats were anaesthetized with ether, and the lower extremity was grasped with the finger tips above and below the knee joint. A firm adduction, or varus, force was applied until a definite snap was felt. Occasionally, if the extremity was grasped a few millimeters too low, fractures would occur through the proximal tibial epiphysis. While the animal was still anaesthetized, roentgenograms were made with the extremity in the deformed position (Figs. 3-A and 3-B). The deformity was then reduced, and the animal replaced in the cage. On a few occasions fractures were produced—as proved later by gross and microscopic studies—which were not apparent on the original roentgenograms because the fracture had slipped back into place before the films were made. This situation has been encountered clinically, and such a spontaneously reduced Type I fracture can be easily overlooked.

No attempt was made to immobilize the extremity. The animals were sacrificed at intervals of from immediately after the injury to fifty-two days, and roentgenograms were again made. In some cases both lower extremities were used at different times; in others, the normal extremity served as a control. The bones were then fixed in formalin and decalcified, and paraffin sections were made. The femora were cut in the frontal plane, and representative sections were taken from the anterior, middle, and posterior parts of the bone. This was found to be worth while since the healing process seen in the anterior portions did not always correspond to that found at deeper levels.

RESULTS

First Week

At the time of injury a rupture of the lateral periosteum occurs (Fig. 4). This tear takes place a few millimeters above the epiphyseal line, leaving a tag of periosteum attached to the epiphysis. This tag was found trapped within the fracture line in several animals (Fig. 5). The varus angulation and medial displacement of the epiphysis result in elevation, but not in tearing, of the medial periosteum. Subperiosteal hemorrhage occurs, and later in the first week hyperplasia of the osteogenic cells is noted beneath this raised periosteum. The fracture line itself was found to enter the lateral, or distraction, side of the epiphyseal plate through the hypertrophic zone. The distal femoral epiphyseal line in rats, as well as in human beings, is peaked at its center. This peak, or apex, seems to be vulner-

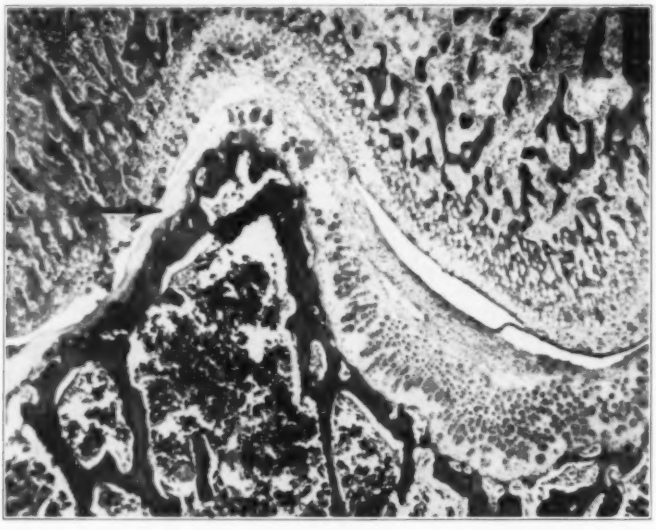

FIG. 6

Photomicrograph, showing fracture line forty-eight hours after injury. The fracture passes through the hypertrophic zone of cells on the distraction side but involves deeper layers near the apex. On the compression side of the apex is an area of cartilage necrosis (arrow).

able to the trauma of a Type I fracture. As the fracture defect is traced medially it is noted that it descends deeper into the epiphyseal cartilage and in most of the animals involves the resting layer of cells at the apex, or center, of the epiphyseal cartilage (Fig. 6). In some, even the subepiphyseal cartilage bone plate was injured at this central point. Since the apex is more prominent anteriorly, apical injury was not as commonly seen in the histological sections procured more posteriorly. On the medial, or compression, side, the fracture line continued for a short distance in the deeper layers of cartilage and then made its exit to the medial periosteum through either the zone of hypertrophic cells or the metaphyseal bone, leaving a small triangular metaphyseal fragment attached to the epiphyseal cartilage. Where the metaphyseal fragment was present, it seemed to have offered protection to the normal-appearing epiphyseal cartilage distal to it. In medial areas where there was no metaphyseal fragment, the epiphyseal cartilage frequently showed evidence of significant injury. This was particularly common in the region just medial to the apex at the center of the epiphyseal plate. Here, areas of necrosis of the entire depth of the plate were encountered; the cells in the region showed little or no staining ability and no evidence of nuclear material. It was believed that these areas had been subjected to the grinding force mentioned

earlier. In most instances, these severely damaged cells were limited to a small area, with normal epiphyseal cartilage on each side. The fracture line itself was found to contain cellular debris and blood. Since, however, most of the fracture line passed through avascular tissue, the amount of hemorrhage present was limited and more marked at the periphery. Strands of fibrin were prominent, and, as these were compressed within the fracture during the healing process, they formed a persistent band which will be referred to as the fibrin line.

Second Week

By the second week, external callus was obvious and the end of the femur had a somewhat bulbous appearance. Subperiosteal new-bone formation was most prominent on the medial side (Figs. 7-A and 7-B). This bone was present along

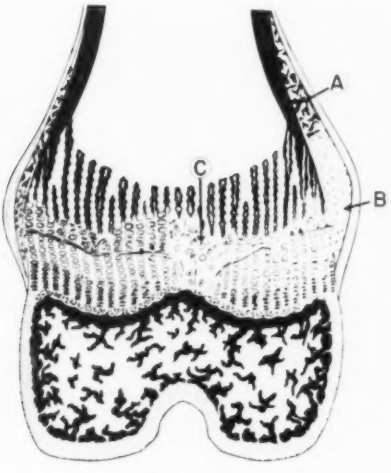

FIG. 7-A

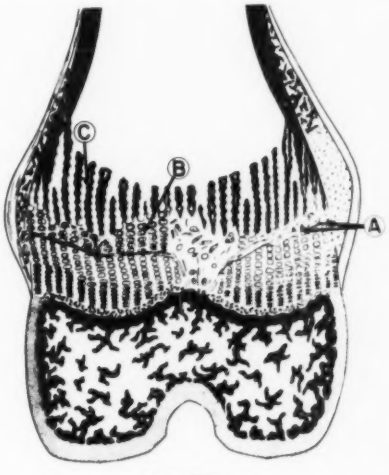

FIG. 7-B

Fig. 7-A: Fracture at eight days: *A*, new subperiosteal bone, more marked on the compression side; *B*, new cartilage beneath the periosteum near the epiphyseal line; and *C*, defect in the epiphyseal plate in the apical area where deeper layers of cells were injured.

Fig. 7-B: Fracture at eight days: *A*, fibrin line marking old fracture site; *B*, layer of cartilage cells remaining on the metaphyseal side of the fracture since the time of injury; and *C*, metaphyseal trabeculae which, along with cartilage and fibrin, are undergoing resorption.

the shaft but absent at the level of the epiphyseal plate where only cartilage cells could be found. The fracture line was clearly marked by the band of fibrin. The reaction which occurs about the cartilage cells remaining on the metaphyseal side of the fracture is of interest. It might be anticipated that these few cells would be used up in the normal process of enchondral ossification, leaving metaphyseal trabeculae adjacent to the fracture line. This probably does occur to a limited extent when only a few cells of the zone of provisional calcification are proximal to the fracture. However, when a larger segment of cartilage is present on the metaphyseal side, there is interference with enchondral ossification. The tips of the metaphyseal trabeculae immediately adjacent to this cartilage undergo resorption, leaving a space in which appear vascular connective tissue and giant cells. Under these circumstances the cartilage proximal to the fracture line on the metaphyseal side is not immediately removed but may persist for several weeks. The cartilage cells distal to the fracture line continue to proliferate but the normal invasion by vascular tissue and enchondral formation of bone does not occur, since the metaphyseal circulation is blocked by the line of fibrin and proximal cartilage and cannot reach the cartilage distal to the fracture. This leads to widening of the

epiphyseal plate (Fig. 8). Toward the end of the second week, in areas where the proximal cartilage and fibrin debris have been removed from the fracture line, the metaphyseal circulation reaches the widened epiphyseal cartilage distal to the fracture line, and the normal process of enchondral ossification is resumed. This, then, is the first process of healing, which seems to occur where the fracture line is proximal in the zone of hypertrophic cells. This has been described by Dale and Harris.

Third Week

In the majority of animals studied, however, healing by means of this first method did not occur. By the third week the cartilage cells on the metaphyseal side of the fracture were still in evidence as was the fibrin line marking the fracture

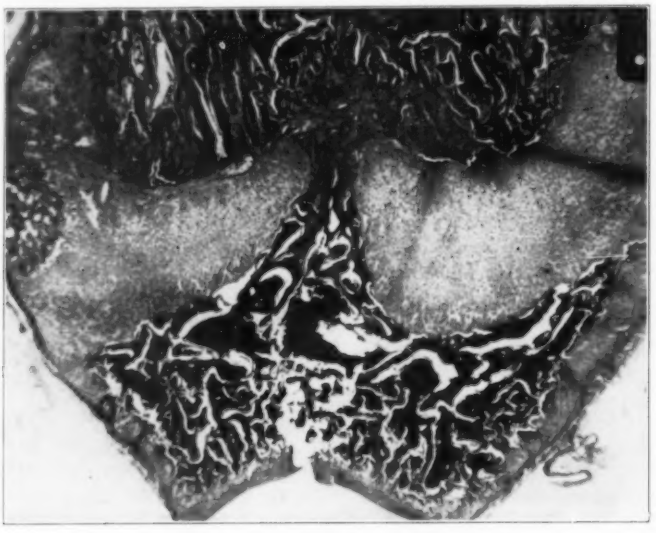

FIG. 8

Photomicrograph of the distal end of a rat's femur eight days after fracture. Note the marked widening of the epiphyseal plate on either side of the central apical defect.

site (Fig. 9). However, these cartilage cells had been further displaced away from the epiphysis by the marked widening of the portion of the epiphyseal plate distal to the fracture. The maturation of the cartilage cells in this widened portion of the plate was of particular interest because those cells next to the fibrin line, and therefore farthermost from the resting zone, were not the most mature. On the contrary, the cells lying equidistant between the fracture line and the resting zone showed signs of greatest maturity. With further growth this zone of mature cartilage cells was invaded by vascular tissue, thereby splitting this portion of the epiphyseal plate into proximal and distal layers. This vascular tissue originated from the periphery, from the apical defect, or from other defects in the proximal portion of the plate. With the ingrowth of this vascular tissue into the mature cartilage cells, enchondral ossification began in the middle of this widened epiphyseal plate (Fig. 10). As this ossification center enlarged, bone formation in the distal cartilage proceeded in the normal manner. However, in the proximal layer of cartilage, with its base on the fibrin line, bone formation progressed in the reverse direction. As a result, the sections of the healing epiphyseal plate at this stage show a normal-appearing epiphyseal plate with normal enchondral bone formation, but proximal to this is another cartilage plate functioning in a reverse

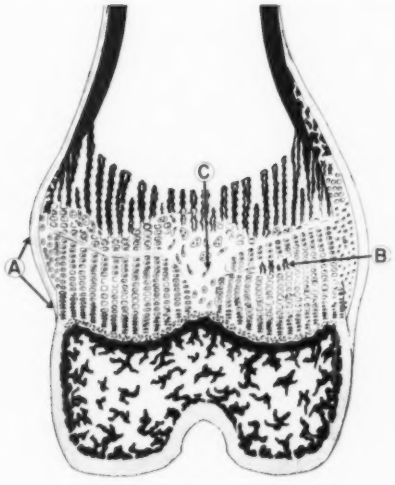

FIG. 9

FIG. 10

Fig. 9: Fracture at fourteen days: *A*, marked widening of the epiphyseal plate due to failure of the metaphyseal circulation to reach cartilage cells distal to the fracture line; *B*, ingrowth of vascular tissue to the center of the widened epiphyseal plate, maturation of central cartilage cells, and early new enchondral bone formation; and *C*, apical defect, not quite so wide as at earlier stage shown in Fig. 7-A.

Fig. 10: Fracture at twenty days: *A*, still further widening of epiphyseal plate; *B*, new enlarging ossification center in the middle of the wide epiphyseal plate with enchondral ossification occurring from the distal and proximal cartilage layers; *C*, further narrowing of the apical defect by crowding from the adjacent epiphyseal cartilage; and *D*, cartilage proximal to the fracture line undergoing resorption in the presence of giant cells and granulation tissue.

FIG. 11-A

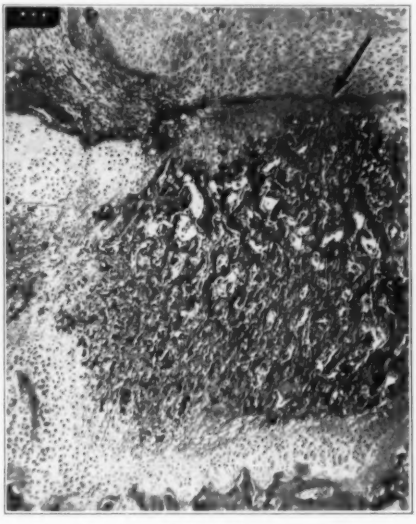

FIG. 11-B

Fig. 11-A: Photomicrograph of section, twenty days after fracture, showing the new ossification center with cartilage distal and proximal to it. There is also a wide layer of cartilage on the metaphyseal side of the fracture which is involved in enchondral ossification.

Fig. 11-B: Higher magnification of the area enclosed in the rectangle in Fig. 11-A. Arrow points to the fibrin line of the old fracture site.

direction. Still farther proximally is the fibrin line of the old fracture site, and on the metaphyseal side of this line is still a third layer of cartilage—that remaining with the metaphysis at the time of fracture (Fig. 11-A). This, for the most part, is not functioning, but in a very few sections thicker parts of this layer of cartilage did seem to be taking part in enchondral bone formation. The picture of a triple epiphyseal line was thus created (Fig. 11-B). This splitting of the wide epiphyseal plate and formation of a new ossification center is a second process of healing.

Both the first and second healing processes were observed in various parts of the plate, but in areas of greater damage, such as at the apex, still a third method was observed. Varying degrees of injury to the apical region were noted. In some instances the trauma had apparently been sufficiently great to break through the subepiphyseal cartilage bone plate. Where this occurred, there was communication

FIG. 12

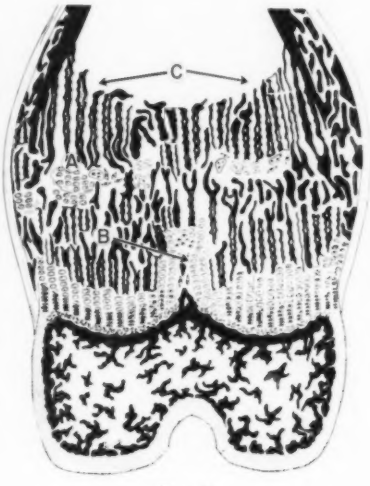

FIG. 13

Fig. 12: Photomicrograph thirty-four days after fracture, showing further widening of the ossification center.

Fig. 13: Fracture at thirty-six days: *A*, resorption of the proximal cartilage layers, leaving only scattered islands of cartilage cells and permitting communication between the metaphyseal bone and the new ossification center; *B*, fibrous strands and irregularity in epiphyseal plate mark the remains of the apical defect; and *C*, persistence of metaphyseal trabeculae results in some sclerosis.

at the apex between the marrow cavities of the epiphysis and metaphysis. Usually, this amount of damage involved only a small area of the epiphyseal plate, and sections taken at different levels showed regions of less severe apical injury. The defect remaining in the epiphyseal plate was usually filled with fibrous tissue and scattered cartilage cells. The healing of these defects, the third healing process, came about through the proliferation of these few remaining cartilage cells at the base of the defect and by the encroachment, from all sides, by the adjacent normal epiphyseal cartilage (Figs. 7-A, 9, and 10,*C*).

Fourth to Sixth Weeks

By the fourth week there was little evidence of the old injury in those femora which had apparently healed by the first process—that is, by resorption of both the fibrin line and the proximal cartilage with resumption of normal enchondral ossification. Eight of the twelve animals in this group, however, demonstrated at least partial healing by the second process. Here there was still obvious evidence of the split epiphyseal plate. The ossification center between the layers of the split

plate had widened considerably (Fig. 12). By the fifth week, resorption of portions of the proximal cartilage layers and the fibrin line had taken place, and, later, only a few scattered islands of cartilage were to be found in the metaphyseal region (Figs. 13 and 14).

During the healing process the normal remodeling of the metaphysis is impaired for, as growth takes place, the metaphysis remains as wide as the epiphyseal plate, giving it a flask-shaped appearance. There is also delay in the resorption of the intramedullary metaphyseal trabeculae, resulting in some sclerosis of this region, as seen by roentgenography.

FIG. 14

Photomicrograph of fracture at thirty-six days shows changes noted in Fig. 13.

Growth measurements were not made on all of the animals in this series. Of those studied there was no instance of overgrowth due to the epiphyseal fracture. Definite retardation of growth was found in those fractures that resulted in extensive damage to the deeper layers of the epiphyseal cartilage or to the subepiphyseal cartilage bone plate. In most instances, where healing was proceeding by either the first or second methods, growth retardation amounted to about one millimeter in animals sacrificed between eight and fifty-two days; this small degree of shortening did not correlate with the length of time between fracture and sacrifice of the animal.

DISCUSSION

It is suggested in these studies that the Type I fracture in rats may result in varying degrees of damage to the epiphyseal plate. The direction taken by the fracture line through the plate is dependent on several factors. Where distracting forces are involved the fracture occurs in the zone of hypertrophic cells. Compression results in a fracture through the metaphyseal trabeculae. A combination of shearing and compression may result in a grinding of the metaphyseal bone into the epiphyseal plate, damaging all layers of cells. In addition, the configuration of

a particular portion of the epiphyseal plate, such as that of the apical region, may make this part more susceptible to injury. It is also suggested that the level of the injury to the epiphyseal plate determines its healing process. If the fracture is proximal in the hypertrophic zone, healing takes place by resorption of the proximal cartilage and fracture debris, a process which temporarily delays enchondral bone formation and causes moderate widening of the epiphyseal plate. After resorption of the proximal cartilage and debris, normal enchondral ossification is resumed. This is usually complete by the end of the third week. If the fracture is more distal or deeper in the hypertrophic zone, considerable delay in resorption of the proximal cartilage is encountered. Meanwhile, marked widening of the epiphyseal plate distal to the fracture occurs. The cells in the middle of this wide plate mature and are invaded by vascular tissue. This results in a split epiphyseal line—a second healing process—which may not be completed until after the fifth week. A third healing process concerns defects caused by injuries involving the entire depth of the plate. This involves either hyperplasia of the few scattered remaining cartilage cells of the resting zone or narrowing of the defect by encroachment of the normal epiphyseal cartilage on all sides. This third process is of limited capacity. If the defect is large, healing cannot take place, and growth in that portion of the epiphysis ceases.

SUMMARY

Fractures through the distal femoral epiphysis of rats were produced by applying a varus angulating force. This resulted in distraction on the lateral side of the epiphyseal plate and compression on the medial side. On the distraction side the fracture line passed through the zone of hypertrophic cartilage cells. On the compression side it either involved the metaphyseal bone or resulted in a grinding injury to deeper layers of epiphyseal cartilage cells. Three types of healing are described which are determined, apparently, by the level of the fracture within the epiphyseal plate.

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DISCUSSION

Dr. Alexander P. Aitken, Brookline, Massachusetts: The common avulsion type of epiphyseal injury, which I have chosen to call Type I, does not lend itself to scientific investigation in human beings. Consequently, little is known of the exact level of fracture. I have assumed and others have demonstrated that the fracture line passes through the zone of degenerating or hypertrophic cells and does not involve the all-important rest or storage zone. Hence, the rarity of gross disturbances in this type of injury in human beings.

The epiphysis, as described by Dr. Brashear, is usually pulled off on one side by the attached ligaments and is associated with a fracture of the metaphysis on the opposite side, due to a compression force. Dr. Brashear states that the size of the metaphyseal fracture seems to depend on the magnitude of the compression force.

In human beings, it appears to depend more on the relationship of the thickness of the epiphyseal plate to the width of the shaft. For only in the very young person, when the zone of epiphyseal cartilage is wider in relationship to the shaft, de we see true epiphyseal fractures without fractures of the metaphysis.

In older children, when the zone of cartilage is relatively narrow, we see less involvement of the epiphysis with larger metaphyseal fragments. It could be argued that in the young person this phenomenon is due to the greater cushioning effect of the cartilage against com-

(continued on page 1076)

Preliminary Findings from a Roentgenographic Study of the Influence of Heel Height and Empirical Shank Curvature on Osteo-articular Relationships in the Normal Female Foot

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This investigation is based on a study of successive lateral roentgenograms of selected subjects made under carefully standardized conditions to facilitate direct comparative measurements with minimum error. Great care was taken to ensure the highest possible degree of uniformity in the placement of the feet with respect to the tube center. A specially prepared platform provided support for the cassette and enabled the subject to stand at ease with weight well distributed between the right and left foot. The center ray was directed at a point directly under the lateral malleolus and mid-way between the malleolus and the platform (Fig. 1).

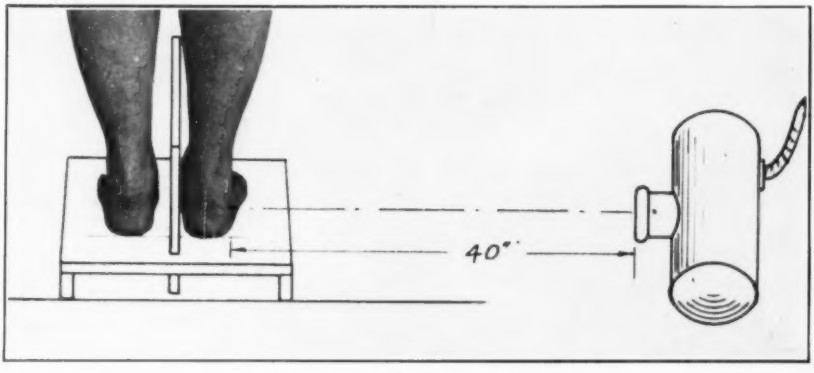

FIG. 1

Schematic illustration of technique used in making successive exposures.

Initial explorations were conducted on two subjects whose feet were of similar over-all length, but which revealed individual differences within a range generally described as structurally normal. Subject A wore size 7AA shoes. Her feet were slender with a very high longitudinal arch, approaching a mild cavus. The subject revealed moderately strong pronation in both stance and walking. Subject B wore size 7½ A shoes. The well developed medial longitudinal arch was not excessively high, and this subject revealed only slight pronation when standing and walking. Simplified tracings from roentgenographic comparisons of the right feet of respective subjects are illustrated in Figure 2.

The twofold purpose of this investigation has necessarily influenced the data to be sought and the reference levels chosen. Our first concern is the recording and presentation of information that will contribute to the general knowledge and understanding of the function of the human foot and, therefore, of its functional needs. Secondly, since footwear profoundly affects the continuing well being of

the foot, it is vitally important that shoe lasts be designed to meet, in the fullest measure possible, the requirements of the foot in action.

It is generally believed that feet function somewhat differently in shoes, and particularly in shoes of different heel heights, than they do barefoot. To reveal any such differences the functional posture of each foot was recorded roentgenographically under three sets of conditions:

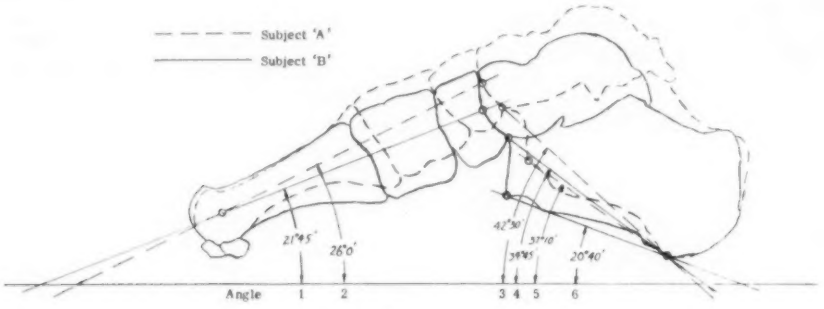

FIG. 2

Tracings of comparative lateral roentgenograms of the right foot of subjects used.
Fore part of the foot: Angles 1 and 2 formed by intersection of line connecting center of first metatarsal head and center of head of the talus with the floor line.
Lateral longitudinal arch: Angles 3 and 4 formed by the intersection of a line connecting the mid-point of the plantar aspect of the calcaneal tuberosity and the plantar aspect of the calcaneocuboid articulation with the plane of the floor.
Medial longitudinal arch: Angles 5 and 6 formed by intersection of a line connecting the mid-point of the calcaneal tuberosity and the superior surface of the anterior lip of the calcaneus at the calcaneotalar articulation.

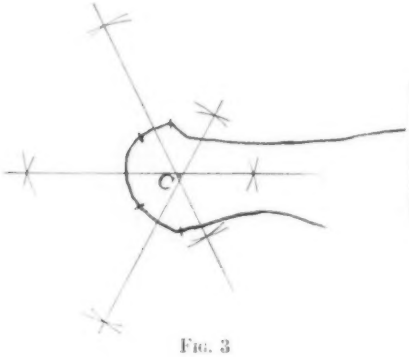

FIG. 3

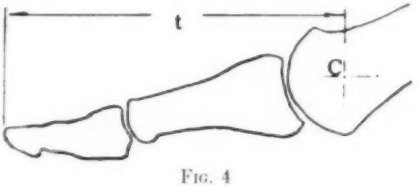

FIG. 4

Fig. 3: Geometry tells us that the unknown center of any circle (or arc) can be found by locating three points on the circle and bisecting the distances between points 1 and 2, and 2 and 3. The center is at the point of intersection of the two bisecting lines. Since the plane view of the first metatarsal head is a slightly irregular arc, four points were used, resulting in three bisecting lines which formed a small triangle enclosing the hypothetical center of the head. Point C' is located at the center of this triangle.
Fig. 4: Measurement of horizontal distance (t).

Series 1: With the subject standing at ease barefoot and in shoes of five different heel heights.

Series 2: With the subject standing barefoot, but with the heel of the foot supported only by shoe heels corresponding to those on the shoes used in Series 1.

Series 3: With the subject standing barefoot with the heels elevated by voluntary muscle contraction to heights equivalent to the heel heights used in Series 1 and 2. Immediately after each exposure in Series 2, the subject was asked to re-

TABLE I

LENGTH OF HORIZONTAL DISTANCE (t) IN MILLIMETERS

Heel Elevation (*Inches*)	Wearing Shoes	Shoe Heels Only	
		With Weight-Bearing	No Weight-Bearing
	Subject A		
0		65.0	65.0
6/8	65.5	65.0	64.5
9/8	65.5	65.0	64.0
12/8	66.0	65.0	65.0
17/8	66.0	64.5	65.5
21/8	65.5	64.5	65.0
Average	65.7	64.8	64.8
Range	0.5	0.5	1.0
	Subject B		
0	67.0	65.7	65.7
5/8	67.2	65.7	66.0
12/8	67.2	65.5	66.0
13/8	66.7	65.5	66.0
13/8	67.0		
14/8	67.0	65.7	65.7
17/8	66.7	65.5	65.7
Average	67.0	65.6	65.8
Range	0.5	0.2	0.3

move body weight from the heels, without removing the foot from light contact with the shoe heel, while a second exposure was made.

Our primary reference point C' (Fig. 3) was selected because (1) it has long been recognized as a point of significance in any consideration of foot function, (2) it is one of the most important points used in the determination of correct shoe fitting, (3) its location in the foot is well suited to the geometric constructions to be made, and (4) its small size, relatively regular shape, and proximity to the underlying weight-bearing surface minimize errors in measurement.

Six reference points were selected on the basis of the significance of respective areas in relation to the information sought:

1. First metatarsal-head center point;
2. Mid-point of the vertical plane of the distal surface of the head of the talus;
3. Plantar aspect of the calcaneocuboid articulation;
4. The superior surface of the anterior lip of the calcaneus at the calcaneotalar articulation.
5. The approximate mid-point on the plantar aspect of the calcaneal tuberosity;
6. The posterior limit of the calcaneal tuberosity.

Each of these was carefully located on the roentgenogram of the respective feet in barefoot stance with no heel elevation. Successive roentgenograms of the feet at increasing heel elevations were laid over the transilluminated barefoot roentgenogram with respective reference areas exactly superimposed and the location of reference points marked on each film. By this procedure the location of the reference point relative to the particular bone on which it was located was kept constant in the presence of multiple changes in the relationships among the bones of the foot.

Attention was first directed to the definition of changes in length of the great toe, measured from the center of the first metatarsal head to the tip of the distal phalanx, accompanying changes in heel elevation. The method of measurement is illustrated in Figure 4; data obtained are presented in Table I.

From the data shown in Table I it seems clear that:

1. In the two subjects the differences in length (t) in the heel-height range of from 0 to 21/8 inches does not exceed 1.0 millimeter.

2. Since the differences shown fail to correlate with heel elevation, they do not appear to result directly from heel elevation.

3. Their most probable causes are random differences in toe position and error in measurement.

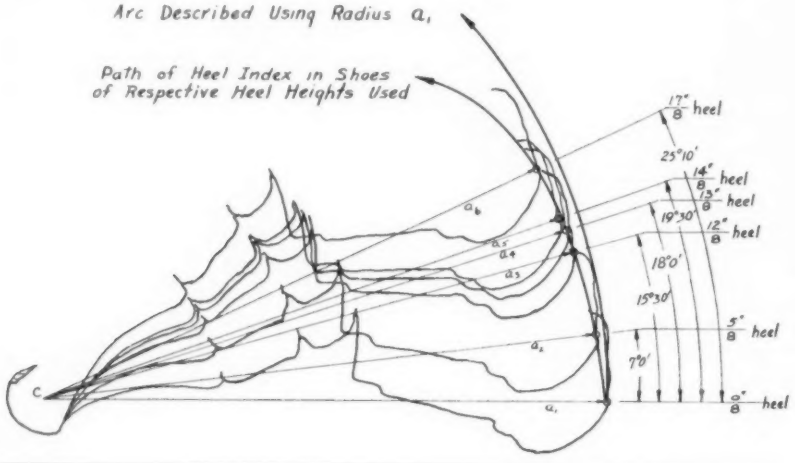

FIG. 5

Tracings of the plantar outline of the skeletal structure of the right foot of subject B standing barefoot and in shoes ranging from 6/8 inch to 17/8 inches height of the shoe heel.

The measurements in Table I indicate that the average length of t (Fig. 4) is slightly less for both subjects when no shoes are worn. Tension on the plantar flexors of the toes to improve stability in unaccustomed barefoot stance on high heels could account for this difference of 1.4 millimeters or less.

A more direct relationship between heel height and the functional posture of the foot is seen in comparisons of the important heel-to-ball distance.

The correct location of the ball point C of the foot in a shoe is simultaneously a requirement for functional efficiency and an index of correct length fitting. It, therefore, becomes a matter of prime importance that the design of the shoe and the shoe last for respective heel heights correspond as closely as possible with changes in structural and functional relationships within the foot under the influence of increasing heel height. Figure 5 clearly reveals the progressive shortening of the heel-to-ball distance of subject B (lines a_2-a_6) accompanying the increase in heel height of successive pairs of shoes worn, compared with line a_1, representing the heel-to-ball length of the unshod foot. Dimensions corresponding to differences seen in lines a_1-a_6 are presented for both subjects in Table II. The numerical values of lines a_1-a_6 were obtained by direct measuremnt from respective roentgenograms and by calculation from values of respective angles formed at C and heel height, using the equation $\sin C = \frac{\text{side opposite C}}{\text{hypotenuse}}$. Since differences in shoe construction, particularly at the heel-seat level, produce a slightly different elevation of the heel of the foot even though the outside heel heights may be the same, the actual elevation of a fixed point on the calcaneus from the floor line was measured on respective plates for increased accuracy.

Question may be raised by the inclusion of both measured and calculated values for a_1-a_6, presented in Tables II and IV. Calculated values were included

TABLE II

CORRELATION OF HEEL-TO-BALL LENGTH OF FOOT WITH SHOE-HEEL HEIGHT*

Subject	External Height of Shoe Heel (*Inches*) A	B	Elevation of Calcaneal Reference Point (*Millimeters*) A	B	Angle Formed at C A	B	Lengths of a_1–a_6 (*Millimeters*) Calculated A	Measured A	Calculated B	Measured B
	Barefoot		3.7	0.0	1° 15′	0° 0′	170.5	171.0	169.6	171.0
	6/8	5/8	23.5	20.5	8° 5′	7° 0′	168.2	168.5	167.1	168.2
	9/8	12/8	44.0	44.5	15° 15′	15° 30′	167.5	166.7	167.3	166.5
	12/8	13/8	58.5	51.5	20° 45′	18° 0′	166.0	166.5	161.5	166.6
	17/8	14/8	65.5	55.0	23° 30′	19° 30′	165.0	165.0	164.3	164.7
	21/8	17/8	93.0	70.0	36° 0′	25° 10′	158.0	164.5	158.2	164.6
Range	21/8	17/8	89.3	70.0	35° 15′	25° 10′	11.5	6.5	11.4	6.4

*It is gratifying to note that, whereas the numerical values for respective subjects wearing different shoes do show expected differences, there is clear evidence of a consistent pattern of behavior under the influence of increasing heel height of shoes worn.

TABLE III

COMPARISONS OF THE MEASURED HEEL-TO-BALL LENGTH OF THE SAME FEET AT INCREASING HEEL ELEVATIONS WITH SHOES AND STANDING ON SHOE HEELS ONLY

Length of a_1-a_6 in Millimeters

Heel Height (*Inches*)	In Shoes	Heels Only	Difference	Heel Height (*Inches*)	In Shoes	Heels Only	Difference
	Subject A, with Weight-Bearing				Subject B, with Weight-Bearing		
0	170.5	170.5	0.0	0	171.0	171.0	0.0
6/8	168.2	169.0	0.8	5/8	168.5	169.7	1.2
9/8	167.5	168.0	0.5	12/8	166.7	170.5	3.8
12/8	166.0	167.7	1.7	13/8	166.5	170.2	3.7
17/8	165.0	167.7	2.7	14/8	165.0	170.4	5.4
21/8	158.0	166.0	8.0	17/8	164.5	170.5	6.0
Range	12.5	4.5			6.5	2.5	

because small errors in measurement, due to differences in definition and contrast, are difficult to eliminate when measurements are made directly from roentgenograms. Since, in this instance, we were able to measure directly all angles and sides of the triangles formed, we had the opportunity to gauge the accuracy of our measuring technique. Clearly, if the measurement of the angles and sides used to calculate a_1-a_6 had been significantly in error, respective values presented in Table II would have shown greater disparity. Furthermore, in our continuing efforts to derive additional data from these roentgenograms we anticipate the possibility that we may be forced to rely on calculated values for data not directly measurable. It is helpful to us to have this index of the potential accuracy of such calculations.

Obviously, heel height is only one of many variations found in women's shoes. Shoes of the same heel height may contain shanks of widely differing curvatures, whereas shoes of different heel height may use shanks of similar curvature. It was clear, therefore, that this exploration should include efforts to differentiate between changes in foot posture due, primarily, to heel elevation and to those arising from other factors. A series of comparisons was conducted with the subjects standing barefoot on shoe heels only of heights corresponding to those of respective pairs of shoes previously worn. Figures 6-A and 6-B present tracings of essential portions of the foot from roentgenograms made under the conditions stated.

The amount of heel-to-ball shortening in shoes, defined in Table II and illustrated in Figure 5, appears to be significantly reduced when the subject stands

barefoot (Series 2) on heels corresponding to the heel heights of shoes worn in Series 1. Figure 6 presents pertinent portions of tracings of the heel area of the respective feet at successive elevations for comparison with Figure 5. Subject A (Fig. 6-A) reveals heel-to-ball shortening to a lesser degree barefoot than was recorded in shoes. Subject B (Fig. 6-B) shows essentially no shortening barefoot in contrast with shortening of 6.5 millimeters in shoes. Numerical comparisons for both subjects in respective series are given in Table III.

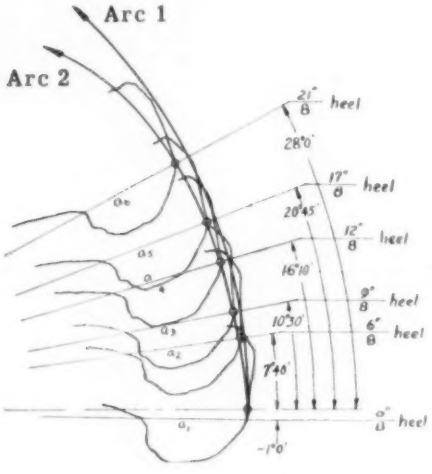

FIG. 6-A

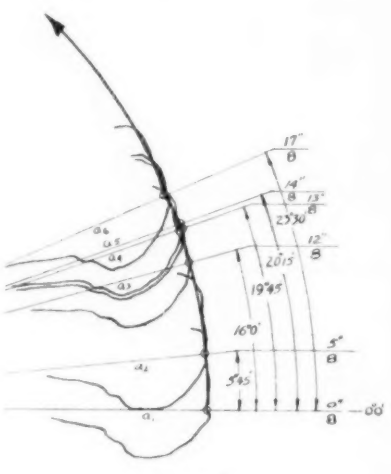

FIG. 6-B

In Fig. 6-A, Arc 1 shows the hypothetical path of the heel index point of subject A at respective heel elevations if no heel-to-ball shortening occurred. Arc 2 reveals the observed heel-to-ball shortening when this subject stood barefoot with the heel raised to the stated elevations. Subject B (Fig. 6-B) revealed essentially no shortening when standing barefoot at the heel elevations stated. Therefore, the hypothetical and observed arcs substantially coincide. Corresponding measurement data are presented in Table III.

In presenting these comparisons, it is recognized that minor differences between the heel angle in shoes and on shoe heels only at respective heel heights do prevail (Figs. 5 and 6-B). These result from the difficulty of relating the unattached shoe heel to the bare foot in precisely the same manner as when shoes were worn. However, the maximum difference between any pair of readings is only 2 degrees, 50 minutes—a variation too small to account for the significant differences found in respective pairs of (a) values for respective subjects (Table III).

Additional interest in the differences just defined is aroused by the close agreement between passive barefoot heel elevation on shoe heels alone, and corresponding active heel elevation through voluntary muscle contraction. The two series of plates (2 and 3) were so similar that it was impossible to make a drawing showing the comparisons. For all practical purposes, the tracings superimposed.

Numerical data, however, clearly define the similarities observed. In Table IV, therefore, are presented both the measured and calculated values of (a) for both subjects at respective heel elevations when:

Series 1. Standing with shoes of respective heel heights;

Series 2. Standing barefoot on shoe heels of corresponding heights;

Series 3: Standing barefoot with the heels elevated to corresponding heights by voluntary muscle contraction.

From the data presented it seems apparent that some characteristic of shoe construction or of the design of the shoe last exerts a progressively shortening effect on the functional posture of the foot as heel height increases. The maximum

TABLE IV

COMPARISON OF HEEL-TO-BALL LENGTH OF THE FEET OF TWO SUBJECTS WITH THE HEELS ELEVATED AS DESCRIBED FOR SERIES 1, 2, AND 3

Heel Height (*Inches*)	Line	Measured Values			Calculated Values		
		Series 1	Series 2	Series 3	Series 1	Series 2	Series 3
Subject A, Heel-to-Ball Length in Millimeters							
0	a_1	170.5	170.5	171.9	169.6	171.9	171.9
6/8	a_2	168.2	169.0	170.8	167.1	168.6	170.8
9/8	a_3	167.5	168.0	168.4	167.3	167.1	168.4
12/8	a_4	166.0	167.7	167.6	165.1	167.0	167.6
17/8	a_5	165.0	167.7	167.7	164.3	167.3	167.7
21/8	a_6	158.0	166.0	165.8	158.2	165.1	165.8
Range		12.5	4.5	6.1	11.4	6.8	6.1
Subject B, Heel-to-Ball Length in Millimeters							
0	a_1	170.5	170.5	170.5	170.5	170.5	170.5
0/8	a_1	170.5	170.5	170.5	170.5	170.5	170.5
5/8	a_2	168.5	169.7	170.0	168.8	169.9	170.3
12/8	a_3	166.7	170.5	170.0	167.1	170.1	169.7
13/8	a_4	166.5	170.2	170.5	166.3	169.7	170.8
14/8	a_5	165.0	170.4	170.2	165.5	170.0	169.9
17/8	a_6	164.5	170.5	170.0	165.2	170.4	169.6
Range		6.0	0.7	0.5	5.5	0.6	0.9

shortening of approximately one-quarter of an inch (six millimeters) reached in both subjects is equivalent to three-quarters of a length size in shoe fitting. Therefore, if the heel-to-ball design of the shoe last does not correspond to the heel-to-ball shortening induced in the foot, significant malplacement of the foot in the shoe can readily occur. Furthermore, whenever the foot becomes displaced longitudinally in the shoe, it is inevitable that it also be displaced with respect to related width characteristics. The resulting functional malrelationships between shoe and foot are most frequently expressed in relation to the fore part of the foot, which is compressed in all directions into the tapering toe outline of the shoe. Acute discomfort, corns and calluses, and in time, deformation of bone structures are the inevitable result. It is, therefore, important that every effort be made to define the mechanism by which the foot is shortened, and the specific areas in which the shortening occurs.

The tracings presented in Figures 7-A and 7-B could have been superimposed in any one of several ways. The method used—superimposing the first metatarsal and cuneiform, thus allowing prevailing differences to be expressed at the heel—has these advantages: (1) It emphasizes the absence of any change in the relationship between the first metatarsal and the first cuneiform. These superimpose so exactly that it is impossible to show two outlines in the tracing. (2) The fixed shape and size of the talus assure maximum constancy in the length of line *AH*. Minor differences shown for this dimension appear to result from slight changes in relationship at the talonavicular articulation.

Geometric constructions superimposed on these tracings provide useful quantitative comparisons. Both subjects reveal approximately 11 degrees of change in the angle formed at the talonavicular articulation (angles A_1 *versus* A_6, Figs. 7-A and 7-B) in the maximum heel-height range used. In addition, it is of interest to note the difference in the numerical value of angle A_1 for respective subjects standing barefoot. The lower medial longitudinal arch of Subject B and the corresponding larger values for angles A_1 and A_6 are associated with the previously described differences in the inherent characteristics of the feet of respective subjects (Fig. 2).

Differences between the heel-to-ball length of the shod and unshod foot throughout the heel-height range most commonly used are quantitatively defined

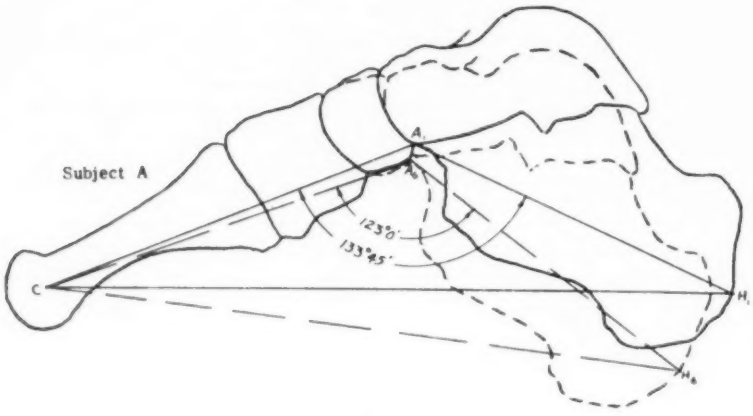

FIG. 7-A

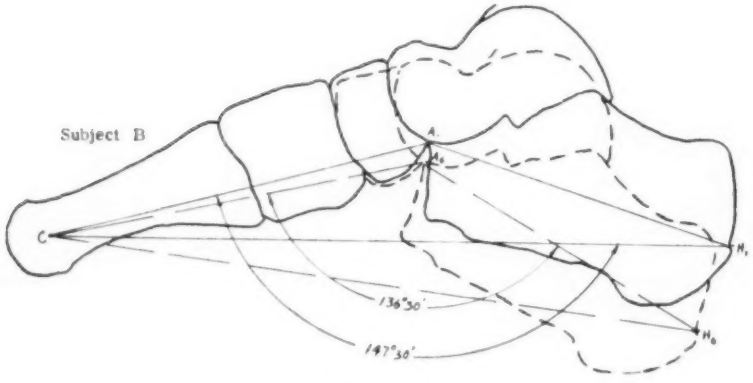

FIG. 7-B

Tracings of the heel-to-ball section of the feet of two subjects showing differences in angulation at the talonavicular and cuneonavicular articulations that accompany increase in heel height from 0 (*H*-1) to 17/8 inches (*H*-6).

in Table IV. That these length changes in the foot result from hinging of the foot, primarily at the cuneonavicular and secondarily at the talonavicular joints, is illustrated in Figures 7-A and 7-B. It, therefore, becomes important to learn why shortening occurs to a greater degree in footwear than barefoot, irrespective of heel height. Figure 8 graphically reveals the reason. Tracings were made of the plantar outline of bones of the foot from calcaneus to first metatarsal head. The four most prominent points on this outline are seen to be (1) the base of the calcaneus, (2) the inferior margin of the calcaneus at the calcaneocuboid articulation, (3) the proximal end of the first metatarsal, and (4) the first metatarsal head. Curves drawn through these points, tangent to respective bone surfaces, provide a measurable index of the shape of the longitudinal arch of the foot under the various conditions defined for Series 1, 2, and 3. The lower curve, tangent to the dotted outline, illustrates the prevailing osseous relationships when the foot is unshod. Whether the heel is passively elevated by stance on a shoe heel (Series 2), or raised by voluntary muscle contraction (Series 3), no demonstrable change is seen in the plantar outline. The upper curve, tangent to the solid outline, clearly indicates a significant change in the relationship of the bones comprising the longitudinal arch. This relative difference was recorded for both subjects when shoes of

increasing heel height were worn. It, therefore, seems apparent that in general the shank curvature used in women's shoes is such that it tends to raise the plantar outline of the longitudinal arch to a height greater than that which the unshod foot would assume at the same heel elevation. From Figures 7-A and 7-B it follows that this increased curvature of the shoe shank is accompanied by a localized hinging of the foot at the cuneonavicular and talonavicular articulations, which result in heel-to-ball length shortening in the foot. The quantitative values for this shortening (Table IV) reveal direct correlation with heel height.

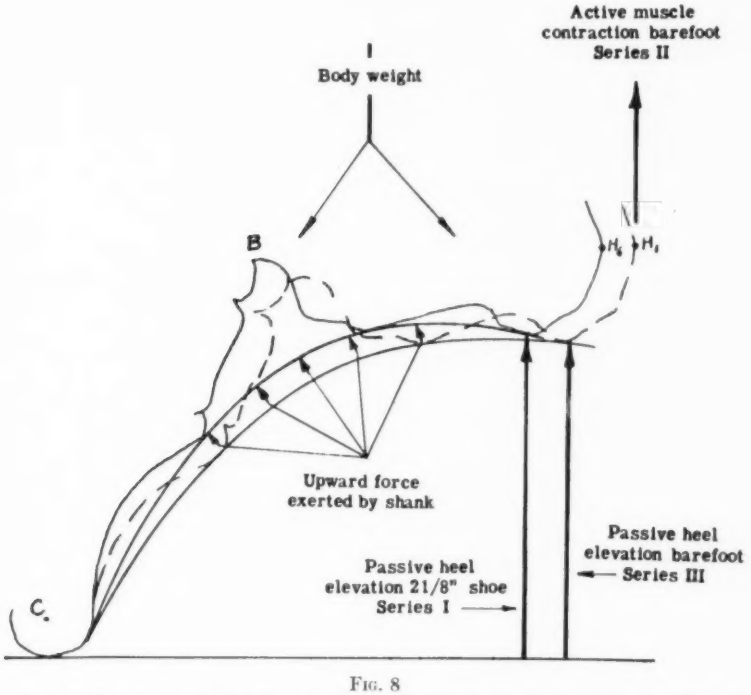

FIG. 8

The solid-line tracing of the foot was made from the roentgenogram of the 21/8 inches heel of Series I, passive stance in a shoe. The broken line represents 21/8 inches heel elevation by both active muscle contraction and passive stance on a 21/8 inches heel (Series II and III, respectively). Only one tracing was used because the outlines of Series II and III were so nearly identical as to be indistinguishable, except in minor detail.

Emphasis has been placed on the empiricism that prevails in relation to the design of shoe lasts. Since the shank must conform closely to the shape of the last, the shank in turn also reflects prevailing empiricism. Unfortunately, the irregular curves expressed in the many shank shapes commonly used have not been defined on the basis of mathematical formulation. To provide some index of the varying shank shapes found in the shoes used in this study, respective curvatures are illustrated in Figure 9.

It has previously been stated that in addition to gaining understanding of the function of the human foot *per se*, it is also our aim to attempt to apply this knowledge to the improvement of the footwear to which feet are subjected. The findings just presented are potentially applicable to the design of shoe lasts.

The last and shoe industries and the medical profession all recognize that the design of shoe lasts is, at present, a mystery. Although technical facilities

for quantity reproduction have substantially improved in reliability, the master model from which production quantities are turned is still designed empirically by individual model makers. These skilled artisans have not had access to quantitative measurement defining the characteristics and requirements of the foot in its constantly changing posture in stance and in walking. They have been required to attempt to express their respective concepts of desirable characteristics of a last in an ever-increasing range of heel heights, more slender outlines, and narrow toes, dictated by frequently changing fashion.

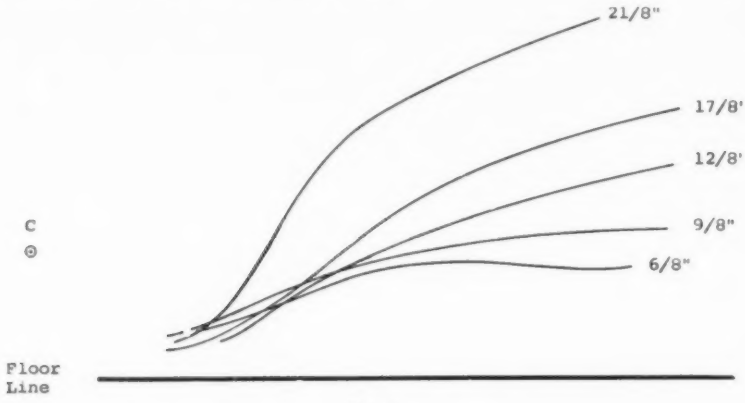

FIG. 9

Graphic comparison of prevailing empirical curvatures, lengths, and positions of shanks used in five shoes worn by subject A, as related to the floor plane and the position of *C*.

We have demonstrated that the heel-to-ball length of the foot is intimately related to the curvature of the shoe shank at all heel heights, and that the amount of shortening can be correlated with the increasing shank curvature required by progressively higher heels. It would, therefore, seem that the best functional fit could be achieved in shoes of any heel height by relating the shank curvature of the last to established requirements of the plantar outline of the bone structure of the foot. It then follows that the heel-to-ball length of such lasts be related to the shortening induced in the foot by the known shank curve used. In short, since heel height, shank curve, and heel-to-ball length are interrelated in the foot, these relationships should be susceptible of formulation and application to the precision design of shoe lasts.

The absence of such correlation, and the continuing empiricism in the design of shoe lasts, undoubtedly account for many of the complaints of improper fit and inadequate support that are directed against present-day footwear. One such complaint, the sliding forward of the foot in the shoe, with resulting poor fit at the heel, failure of mid-foot support, and potential damage to the toes is illustrated in Figure 10. This subject, wearing shoes of several makes, expressing a range of heel heights, reveals the dominant tendency of feet to slide forward in shoes made over lasts expressing arbitrary shank curve and heel-to-ball dimensions. It is our earnest hope that continued research leading to the wider application of precision measurement to the definition of the optimum foot-shoe relationship may, in time, point the way to the elimination of many foot problems arising from the use of improperly proportioned shoes.

CONCLUSIONS

1. A preliminary study has been made of the osteo-articular changes within the foot found in association with (a) increasing heel elevation with and without

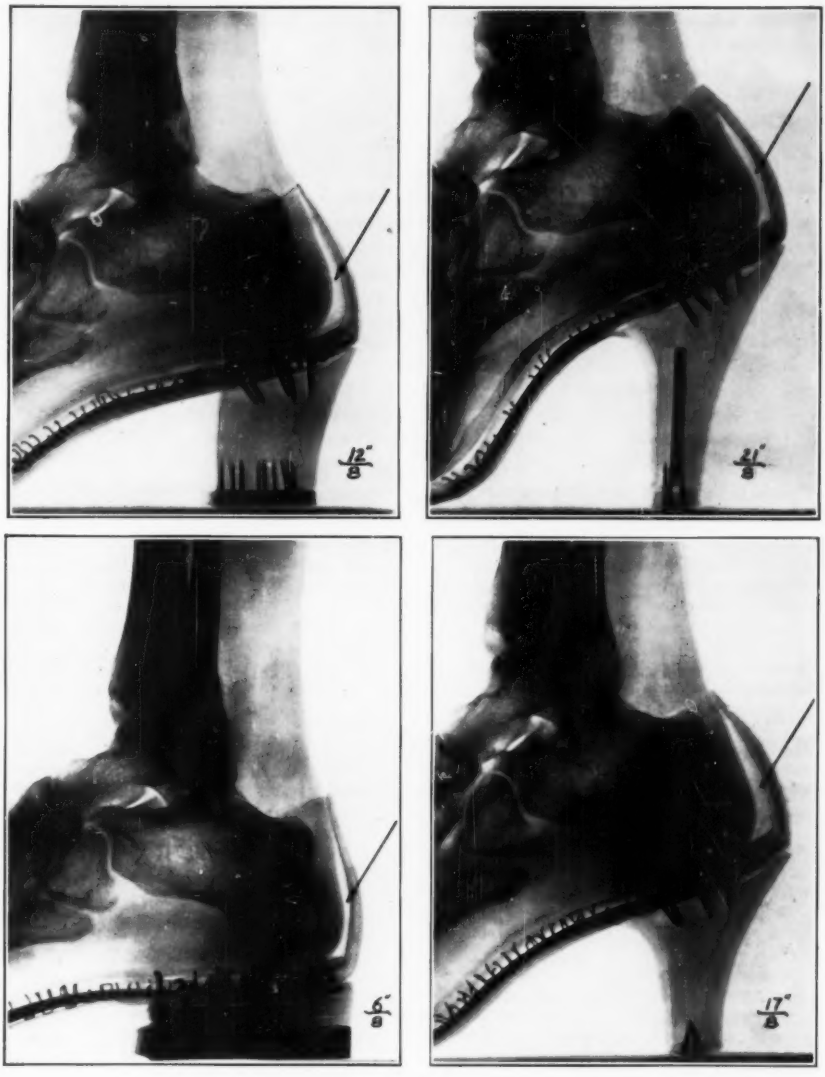

FIG. 10

Examples of malpositioning of the foot in the shoe accompanying elevation of the heel.

shoes and (b) differences in shank curvature found in shoes of different heel heights.

2. Measurements have been made from a series of sixty-five lateral roentgenograms made of the feet of two female subjects under carefully controlled conditions.

3. These measurements strongly suggest that:

a. The length of the foot distal to the first metatarsal head does not change significantly as heel height is increased.

b. The heel-to-ball length of the bare foot may or may not shorten slightly when the subject stands with the heel raised either actively or passively.

c. The shortening of heel-to-ball length of the foot when shoes are worn may increase to twice that observed in the bare foot.

d. This shortening occurs in association with elevation of the longitudinal arch of the foot by the shoe shank, accompanied by a hinging of the foot at the cuneonavicular and talonavicular articulations.

4. The response of normal feet to various heel heights, shank curve, and heel-to-ball relationships should be precisely determined on a mathematical basis.

5. Shoe lasts and shoe shanks should be designed to conform to the relationships found to be required by the foot at respective heel heights.

6. One example is illustrated of the failure of present-day shoe lasts and shoes to meet the needs of the foot in action.

7. We propose to continue these studies for the purpose of attempting to define the specific relationships just indicated on a mathematical basis.

DISCUSSION

Epiphyseal Fractures

(continued from page 1064)

pression, or to the greater violence required to fracture the epiphysis in the adolescent person.

Dr. Brashear described a combination of shear and compression, which may result in grinding of prominent portions of the irregular epiphysis, injuring all zones, especially in markedly displaced Type I fractures, in which the metaphyseal fragment is small. Growth disturbance can, of course, follow any Type I injury in human beings, but the rarity of this disturbance indicates that the distraction or avulsion force is the major one and that compression with damage to the rest or storage zone is relatively unimportant in this type of injury. Our clinical studies also indicate that displacement *per se*, even though extreme, is not a cause of deformity despite the size of the fragment.

Although the roentgenograms in this study indicate that Type I fractures were produced, the findings of damage to the epiphyseal plate were so extensive as to suggest that either the rat epiphysis differs in construction or reacts differently to trauma, or the experimental traumatic force applied was different from that to which human beings are subjected. The described extensive damage to the resting and storage area, even involving the subepiphyseal bone plate, would certainly produce growth disturbance in human beings unless the area was small and was located at a distance from the periphery—the peripheral cortex, I should say. The all-important factor in deformity, of course, in epiphyseal fractures is the relationship of the fracture of the epiphysis to the adjacent cortex.

Dr. Brashear described fragments of cartilage attached to the metaphysis, in many of these cases, due to splitting of the plate, which further suggests a difference in the behavior in the epiphysis of rats when subjected to trauma. When Type I fractures in human beings are operated on cartilage is never found attached to the metaphysis in a true Type I lesion. Healing in human beings would therefore occur by the first process of healing as described by Dr. Brashear. The persistence of cartilage cells on the end of the metaphysis led to the peculiar phenomenon of a triple epiphyseal line, which persisted into the fourth or fifth week, which led to the development of a secondary ossification center between the layers of the split plate, which apparently was visible on roentgenograms. This is an interesting phenomenon and one which I have never seen in my series of epiphyseal fractures in children. Dr. Brashear further describes a third process of healing, when the rest zone and subepiphyseal bone plate were involved, which he describes as being of limited capacity. When the defect was large, growth of the epiphysis ceased, which certainly would have caused premature ossification and deformity, especially if the area was in apposition to the cortex.

I hope Dr. Brashear will continue this study, subjecting another series of rats to this trauma and allowing them to grow to maturity so that the incidence of growth disturbance may be determined. The findings described by Dr. Brashear in rats may occur in human beings, of course, and may account for the premature ossification and retardation of growth and even growth deformity. The fact that growth disturbances in Type I fractures in human beings are rare and usually of little clinical significance would indicate that Type I fractures produced experimentally in animals probably are not comparable in many respects to Type I fractures in human beings. Nevertheless, this has been a most interesting paper and Dr. Brashear is to be commended for the excellence of his work. If he can demonstrate after further study that his rats show as little growth disturbance as human beings, after such extensive damage, then he will have made a real contribution and we will have to revise our thinking.

Transfer of the Long Toe Extensors for Imbalance of the Foot Following Poliomyelitis

BY EUGENE H. CHAPMAN, M.D., PROVO, UTAH

From the Carrie Tingley Hospital for Crippled Children, Truth or Consequences, New Mexico

Because side-to-side balance of the foot must be taken into consideration by the surgeon who is attempting to correct paralytic drop-foot by tendon transfers, the method described here should be considered in conjunction with the method of replacement of the paralyzed tibialis posterior by the peroneus longus described in the excellent article of Fried and Hendel and attributed originally to von Baeyer. The most frequent form of paralysis about the foot and ankle caused by poliomyelitis in patients at Carrie Tingley Hospital has been residual absence or weakness of the anterior and posterior tibial muscles. In Peabody's classification this group of patients corresponds to Group I, limited extensor-invertor insufficiency, and Group II, gross extensor-invertor insufficiency. As a result of study of the series of cases presented in this article, it has become evident that the function of both muscles must be replaced by tendon transfer if satisfactory balance of the foot and ankle is to be achieved without tarsal fusion.

Transfer of long toe extensors in one form or another has been used and described by many surgeons, notably Jones, Hibbs, and Peabody, but the use of toe-extensor transfers to gain active dorsiflexion of the foot has not gained the wide acceptance it deserves, possibly owing to lack of an adequate statement of procedure to guide the surgeon who performs tendon transfers about the foot and ankle only occasionally. The procedure described here is similar to the procedure used extensively by Dr. P. A. Pemberton at the Shriners' Hospital in Salt Lake City and, according to him, it has been in standard use by orthopaedic surgeons in Oklahoma City.

INDICATIONS

By far the most frequent condition for which toe-extensor transfer has been used at Carrie Tingley Hospital has been inadequate or ineffective dorsiflexor power of the foot resulting from poliomyelitis. In most cases, there should be a reasonable prospect that the transfer or multiple tendon transfers will balance the foot and ankle sufficiently to allow the child to go without a brace. Tendon transfers about the foot and ankle are usually not indicated if the child is wearing a toe-to-groin brace for hip and knee weakness. An exception to this rule is exemplified in Case 32 (Table I), in which tendon transfers about the foot and ankle were performed to prevent the progression of an equinus and valgus deformity which could not be controlled by bracing. In such a case, the parents and the patient should be specifically warned that the tendon transfer is being performed to prevent progressing deformity and that the brace will be reapplied at some stage after the surgery.

Toe-extensor transfers for poliomyelitis should not be performed less than two years after the acute attack, in order to allow as much spontaneous recovery of muscle function as possible. Only patients with weakness due to poliomyelitis will be described here. The transfer has also been used at this hospital in an occasional, carefully selected case of cerebral palsy; in club-foot in preference to the Garceau transfer of the anterior tibial; in congenital cavus deformity with claw toes and ineffective dorsiflexion of the foot; and in slowly progressing cases

TABLE I

FOLLOW-UP FINDINGS ON FORTY-FOUR CONSECUTIVE TOE-EXTENSOR TRANSFERS FOR PARALYSIS DUE TO POLIOMYELITIS

Key: AT = anterior tibial; GS = gastrocnemius-soleus; PT = posterior tibial; P = peroneus longus and brevis; EDL = extensor digitorum longus; EHL = extensor hallucis longus; FDL = flexor digitorum longus; FHL = flexor hallucis longus; TA = tendo achillis.

Muscle power: 5 = normal; 4 = good; 3 = fair; 2 = poor; 1 = trace; 0 = absent.

Case and Foot	Preoperative Deformity and Imbalance	Preoperative Muscle Test	Surgical Procedure	Age at Surgery (*Years*)	Follow-up (*Months*)	Activity of Transfer	Residual Deformity	Subsequent Treatment Required
1. L. G. Left	Pes planus; dorsiflexion ineffective on swing phase of gait; toe-extensor substitution on attempted dorsiflexion	AT = 4 GS = 3 PT = 2 P = 4 EDL = 4 FDL = 3 EHL = 4 FHL = 3	5/9/56, transfer of EDL and EHL to medial cuneiform	5	17	Good dorsiflexor power; heel-toe gait	Continuing pes planus	None to date
2. F. C. Left	Toe-heel gait; toe-extensor substitution; pes planovalgus	AT = 0 GS = 4 PT = 0 P = 4 EDL = 5 FDL = 2 EHL = 5 FHL = 2	5/21/56, transfer of EDL and EHL: 3 to medial cuneiform, and 2 to 2nd metatarsal	8½	18	Toe-heel gait; slight dorsiflexor power	Drop-foot and moderate pes planus	Leg brace; mid-tarsal fusion will be needed
3. J. E. Right	Toe-heel gait; pes planovalgus	AT = 0 GS = 5 PT = 0 P = 4 EDL = 5 FDL = 4 EHL = 5 FHL = 4	5/21/56, transfer EDL and EHL to medial cuneiform	5	19	Good dorsiflexor power; gait almost normal	Pes planus	8/14/57, transfer of FDL and FHL to replace PT; slight pes planus still present
4. F. G. Right	Toe-heel gait and pes planovalgus (a)†	AT = 0 GS = 5 PT = 4 P = (a)† EDL = 2 FDL = 5 EHL = 5 FHL = 5	6/1/56, transfer of EDL and EHL to medial cuneiform	12½	21	Good dorsiflexor power; heel-toe gait	Slight pes planus	None
5. L. G. Right	Toe-heel gait; toe-extensor substitution	AT = 1 GS = 4 PT = 3 P = 4 EDL = 3 FDL = 4 EHL = 4 FHL = 4	6/4/56, transfer of EDL to medial cuneiform	5	17	Fair dorsiflexor power; heel-toe gait	Cock-up of great toe on attempted dorsiflexion	4/18/58, transfer of EHL to 3rd metatarsal base; TA lengthening; reefing of EDL transfer; good dorsiflexor activity
6. A. M. M. Right	Tight heel cord; pes valgoplanus; gait not analyzed (flail left leg)	AT = 0 GS = 4 PT = 1 P = 3 EDL = 3 FDL = 4 EHL = 4 FHL = 3	6/6/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform; tendo achillis lengthening	7½	17	Good dorsiflexor power	Moderate pes planus and valgus	3/27/57, Durham flat-foot procedure and transfer of FDL to replace PT; pes planus corrected
7. K. B. S. Right	Toe-heel gait	AT = 1 GS = 2 PT = 0 P = 3 EDL = 2 FDL = 3 EHL = 2 FHL = 4	6/8/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	5½	20	Poor dorsiflexor power; toe-heel gait	Drop-foot	Leg brace required; mid-tarsal fusion and tendon transfer will be required

8. E. E. Right	Toe-heel gait; moderate varus of foot	AT = 3 GS = 4 PT = 4 P = 2 EDL = 4 FDL = 4 EHL = 4 FHL = 4	6/8/56, transfer of EDL to 4th metatarsal base and EHL to 2nd metatarsal base	6½	23	Good dorsiflexor power; heel-toe gait	None	None
9. E. G. Right	Varus after transfer of peroneals to "mid-tarsal" region; poor dorsiflexor power	AT = 0 GS = 3 PT = 4 P = — EDL = 3 FDL = 4 EHL = 3 FHL = 4	6/11/56, transfer of EDL to 4th metatarsal base	11	20	Poor dorsiflexor power	Recurrent equinovarus	4/8/57, transfer of EHL to cuboid; 9/23/57, transfer of PT and FHL to cuboid; position satisfactory
10. G. S. Left	Toe-heel gait	AT = 1 GS = 2 PT = 1 P = 2 EDL = 4 FDL = 4 EHL = 4 FHL = 4	6/15/56, transfer of EDL and EHL to medial cuneiform	7	17	Only slight dorsiflexor activity	Partial foot-drop (tenodesis effect of transfer)	Lambrinudi arthrodesis will be needed
11. F. G. Left	Toe-heel gait; marked pes planovalgus	AT = 1 GS = 5 PT = 4 P = (a)† EDL = 2 FDL = 5 EHL = 5 FHL = 5	6/18/56, transfer of EDL and EHL to medial cuneiform	12½	21	Good dorsiflexor power	Marked pes planovalgus	Durham flat-foot procedure using FDL to replace PT will be required
12. J. L. W. Right	Toe-heel gait; heel-cord tightness	AT = 2 GS = 5 PT = 1 P = 5 EDL = 4 FDL = 3 EHL = 4 FHL = 3	6/22/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	12½	20	Active dorsiflexion to within 5 degrees of neutral; toe-heel gait	Excessive GS strength limits dorsiflexion	Drop-foot night brace to prevent TA tightness is required
13. R. M. Left	Toe-heel gait; moderate heel-cord tightness; pes planovalgus	AT = 0 GS = 4 PT = 2 P = 4 EDL = 4 FDL = 4 EHL = 4 FHL = 4	7/2/56, transfer of EDL, EHL, and peroneus tertius to medial cuneiform	6	20	Good active dorsiflexion; heel-toe gait	Moderate valgus and pes planus; slight heel-cord tightness	1/20/58, transfer of peroneus longus through PT sheath to medial cuneiform to provide invertor power (Fried and Hendel procedure); good result
14. R. B. Right	Toe-heel gait; moderate inversion of foot	AT = 0 GS = 4 PT = 3 P = 3 EDL = 4 FDL = 4 EHL = 4 FHL = 4	7/2/56, transfer of EDL and EHL: 2 to medial cuneiform, and 3 to 3rd metatarsal base	6	17	Poor dorsiflexor power; toe-heel gait	Tendency to inversion of foot	7/12/57, transfer of FDL to lateral cuneiform for dorsiflexor power, resulting in fair dorsiflexor power; brace not required; inversion corrected; slight side-to-side instability; toe-heel gait; mid-tarsal fusion will be required
15. R. M. Right	Tendency to cavus and inversion; slight heel-cord tightness with limited dorsiflexor range	AT = 3 GS = 5 PT = 4 P = 3 EDL = 4 FDL = 4 EHL = 4 FHL = 4	7/6/56, transfer of EDL and EHL: 2 to 2nd metatarsal base, and 3 to 3rd metatarsal base	6	20	Good active dorsiflexor power; tendency to inversion corrected	None	None

TABLE I—*Continued*

Case and Foot	Preoperative Deformity and Imbalance	Preoperative Muscle Test	Surgical Procedure	Age at Surgery (*Years*)	Follow-up (*Months*)	Activity of Transfer	Residual Deformity	Subsequent Treatment Required
16. A. R. Right	Varus deformity and adduction of foot; moderate heel-cord tightness	Not recorded	7/9/56, transfer of EDL to 4th metatarsal base	6	13	Good activity; no recurrence of varus and adduction deformity	Slight pes planus; dorsal bunion of 4th MP joint; not symptomatic	None
17. R. P. Left	Toe-heel gait; heel-cord tightness	AT = 0 GS = 5 PT = 4 P = 5 EDL = 4 FDL = 5 EHL = 4 FHL = 5	7/13/56, transfer of EDL and EHL to medial cuneiform	13	22	Inadequate dorsiflexor range	Tight tendo achillis	6/17/57, transfer of peroneus tertius and longus to medial cuneiform; 3/3/56, tendo achillis lengthening; good dorsiflexor range and power present
18. D. D. Right	Toe-heel gait; moderate heel-cord tightness; slight valgus of foot	AT = 0 GS = 4 PT = 4 P = 4 EDL = 2 FDL = 2 EHL = 2 FHL = 0	7/20/56, transfer of EDL and EHL to medial cuneiform; tendo achillis lengthening	11½	14	Good dorsiflexor activity; normal gait	Slight valgus of foot	None
19. R. N. Left	Toe-heel gait	AT = 1 GS = 4 PT = 4 P = 4 EDL = 5 FDL = 5 EHL = 5 FHL = 5	7/20/56, transfer of EDL and EHL to medial cuneiform	8½	22	Good dorsiflexor activity; heel-toe gait	None	None
20. E. E. Left	Toe-heel gait	AT = 0 GS = 4 PT = 3 P = 4 EDL = 4 FDL = 4 EHL = 4 FHL = 4	7/23/56, transfer of EDL and EHL to medial cuneiform	6½	23	Only slight dorsiflexor activity; toe-heel gait	None	No brace required; patient does well if high-top shoe is worn; further surgery may be required
21. R. S. Left	Toe-heel gait; tight heel cord; pes planus	AT = 0 GS = 3 PT = 1 P = 5 EDL = 4 FDL = 5 EHL = 4 FHL = 5	7/25/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	7	23	Good dorsiflexor power	Moderate pes planus	4/7/58, transfer of FDL and FHL to base of 1st metatarsal to replace PT; tendo achillis lengthening; foot improved
22. E. M. Right	Cavus; calcaneus gait prior to peroneus longus transfer to calcaneus	AT = 1 GS = 2(b)†† PT = 0 P = 4 EDL = 2 FDL = 2 EHL = 4 FHL = 2	7/27/56, transfer of EDL and EHL to medial cuneiform	6	19	Good dorsiflexor power; only fair plantar flexor power	Rather marked valgus	None to date; triple arthrodesis may be required
23. E. C. Left	Toe-heel gait; cavus; slight tightness of heel cord	AT = 0 GS = 4 PT = 4 P = 4 EDL = 5 FDL = 5 EHL = 5 FHL = 5	7/30/56, transfer of EDL and EHL to medial cuneiform	11	16	Normal dorsiflexor power; good heel-toe gait	Tight tendo achillis responding to stretching; cavus improved	None

24. G. L. Left	Toe-heel gait; pes planovalgus; tight heel cord; toe-extensor substitution	AT = 0 GS = 4 PT = 0 P = 4(c)††† EDL = 5 FDL = 5 EHL = 5 FHL = 5	9/7/56, transfer of EDL and EHL to medial cuneiform; tendo achillis lengthening	7	16	Good active dorsiflexion; heel-toe gait	Moderate pes planus	8/26/57, transfer of FDL and FHL to 1st metatarsal base to replace PT; moderate pes planus present
25. R. B. Left	Toe-heel gait in spite of absent GS; valgus of foot	AT = 0 GS = 0 PT = 3 P = 4(b)†† EDL = 4 FDL = 3 EHL = 4 FHL = 3	9/7/56, transfer of EDL and EHL to medial cuneiform	6	15	Over-all balance of foot good; lateral instability on weight-bearing	None	Triple arthrodesis owing to lateral instability may be required
26. W. Y. Right	Inability to heel-walk; weak dorsiflexor power; pes planovalgus	AT = 1 GS = 5 PT = 2 P = 5 EDL = 5 FDL = 2 EHL = 5 FHL = 4	9/19/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	9	3	Good activity (patient satisfied with result and will not return for more follow-up examinations)		None
27. A. C. Left	Toe-heel gait; cavus; toe-extensor substitution	AT = 2 GS = 4 PT = 4 P = 3 EDL = 5 FDL = 3 EHL = 5 FHL = 3	9/24/56, transfer of EDL and EHL: 2 to medial cuneiform, and 3 to base of 3rd metatarsal	8	15	Good dorsiflexor activity; heel-toe gait	None; cavus improved	None
28. C. C. Right	Toe-heel gait; pes planovalgus	AT = 0 GS = 4 PT = 0 P = 4 EDL = 3 FDL = 4 EHL = 3 FHL = 4	9/26/56, transfer of EDL and EHL to medial cuneiform	6½	19	Slight active dorsiflexion; toe-heel gait	Valgus and pes planus	10/18/57, transfer of peroneus longus to medial cuneiform for dorsiflexion and of FDL and FHL to 1st metatarsal to replace PT, resulting in active dorsiflexion; heel-toe gait; slight pes planus still present
29. S. V. Right	Toe-heel gait; calcaneovalgus on weight-bearing	AT = 0 GS = 1 PT = 0 P = 5(b)†† EDL = 4 FDL = 4 EHL = 4 FHL = 4	10/5/56, transfer of EDL and EHL to medial cuneiform	7	12	Good active dorsiflexion; plantar flexion power graded fair	Valgus of foot	5/24/57, transfer of FDL to 1st metatarsal to replace PT; position of foot improved; calcaneus gait
30. C. C. Left	Toe-heel gait; pes planovalgus	AT = 1 GS = 3 PT = 0 P = 4 EDL = 3 FDL = 3 EHL = 3 FHL = 3	10/10/56, transfer of EDL and EHL to medial cuneiform	6½	19	Good active dorsiflexion; heel-toe gait	Valgus and pes planus	10/30/57, transfer of peroneus longus through PT sheath to navicular to replace PT (Fried and Hendel procedure); position improved

TABLE I—*Continued*

Case and Foot	Preoperative Deformity and Imbalance	Preoperative Muscle Test	Surgical Procedure	Age at Surgery (*Years*)	Follow-up (*Months*)	Activity of Transfer	Residual Deformity	Subsequent Treatment Required
31. G. D. Left	Toe-heel gait; marked valgus	AT = 0 GS = 1 PT = 0 P = 3(b)†† EDL = 3 FDL = 4 EHL = 3 FHL = 4	10/19/56, transfer of EDL and EHL to medial cuneiform	10	18	No activity	Marked valgus of foot on weight-bearing; flail foot	12/13/57, pantalar arthrodesis; brace required prior to last surgery
32. A. C. Right	Progressing equinus and valgus deformity not controlled by bracing	AT = 0 GS = 2 PT = 0 P = 4 EDL = 3 FDL = 3 EHL = 3 FHL = 3	10/22/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	8	15	Fair dorsiflexor power; fair balance; moderate eversion controlled by brace (surgery done to control deformity only; toe-to-groin brace still worn)	None	Wears toe-to-groin brace for hip and knee weakness
33. S. A. Left	Toe-heel gait; pes planus on weight-bearing	AT = 0 GS = 4 PT = 4 P = 5 EDL = 5 FDL = 5 EHL = 5 FHL = 5	10/29/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	9½	15	Good active dorsiflexion; slight inversion during swing phase of gait; slight tendency to toe-heel gait	Moderate dorsal bunion, 1st MP joint; slight pes planus on weight-bearing	None
34. G. D. Right	Toe-heel gait; pes planovalgus	AT = 0 GS = 5 PT = 2 P = 5 EDL = 4 FDL = 5 EHL = 4 FHL = 5	11/9/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	10	18	Good active dorsiflexion	Pes planus and moderate valgus	11/27/57, tendo achillis lengthening, transfer of FDL and FHL to replace PT; Durham flat-foot procedure; pes planus corrected; slight inversion
35. M. F. Left	Moderate varus and equinus	AT = 4 GS = 5 PT = 5 P = 4 EDL = 5 FDL = 5 EHL = 5 FHL = 5	11/12/56, transfer of EHL to medial cuneiform and EDL to 4th metatarsal base	10	17	Good active dorsiflexion; varus corrected	Dorsal bunion, 4th MP joint; not symptomatic	None
36. L. H. Right	Toe-extensor substitution partially dorsiflexes; moderate heel-cord tightness	AT = 1 GS = 4 PT = 1 P = 4 EDL = 2 FDL = 3 EHL = 4 FHL = 3	11/23/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	8	13	No activity	Tight tendo achillis and equinus of foot	Refitted with drop-foot brace on 12/3/57
37. M. F. Right	Toe-heel gait; pes planovalgus	AT = 0 GS = 3 PT = 0 P = 4 EDL = 3 FDL = 4 EHL = 3 FHL = 4	11/23/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	10	17	Good active dorsiflexion; heel-toe gait	Moderate valgus and pes planus	8/23/57, transfer of FHL and FDL to base of 1st metatarsal to replace PT; moderate pes planus and valgus still present

38. M. R. V. Left	Toe-heel gait (brace required); cavus; toe-extensor substitution with cock-up deformity of toes	AT = 4 GS = 3 PT = 4 P = 3 EDL = 4 FDL = 3 EHL = 5 FHL = 3	11/26/56, transfer of EHL to medial cuneiform and EDL to 4th metatarsal base	10	5	Good active dorsiflexion; heel-toe gait; cock-up of toes corrected	None; foot well balanced	None
39. A. A. M. Right	Toe-heel gait (brace not required); cavus; toe-extensor substitution with cock-up deformity of toes	AT = 3 GS = 4 PT = 4 P = 4 EDL = 5 FDL = 3 EHL = 5 FHL = 3	11/28/56, transfer of EHL to medial cuneiform and EDL to 4th metatarsal base	14½	17	Good active dorsiflexion (block to dorsiflexion at neutral); position of foot and toes improved	Non-union of 4th metatarsal fracture seen on roentgenogram, 2/18/58; dorsal bunion, 4th MP joint	2/26/58, bone-grafting of 4th metatarsal and attachment of EDL to lateral cuneiform
40. M. H. G. Left	Toe-heel gait (brace required); cavus; moderate inversion; toe-extensor substitution and cock-up deformity	AT = 4 GS = 5 PT = 5 P = 5 EDL = 5 FDL = 5 EHL = 5 FHL = 5	11/30/56, transfer of EDL and EHL: 2 to medial cuneiform, and 3 to 3rd metatarsal base	13	8	Good active dorsiflexion; heel-toe gait	Moderate residual cavus; toe deformities improved	None
41. P. J. O. Right	Toe-heel gait; heel-cord tightness	AT = 0 GS = 4 PT = 2 P = 4 EDL = 3 FDL = 4 EHL = 3 FHL = 4	12/17/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	7	17	Fair active dorsiflexion; dorsiflexion during swing phase improved, but toe-heel gait still present	None	None; mid-tarsal fusion may be required
42. A. M. Right	Toe-heel gait; toe-extensor substitution	AT = 0 GS = 3 PT = 3 P = 5 EDL = 5 FDL = 3 EHL = 5 FHL = 3	12/17/56, transfer of EDL and EHL to medial cuneiform	6½	11	Good active dorsiflexion; moderate toe-heel gait still present	Slight tendency to valgus when foot is dorsiflexed	None
43. M. A. S. Right	Toe-heel gait; pes planovalgus	AT = 0 GS = 4 PT = 1 P = 4 EDL = 3 FDL = 4 EHL = 3 FHL = 4	12/31/56, transfer of EDL, EHL, and peroneus longus to medial cuneiform	7	11	Fair dorsiflexor power; moderate tendency to toe-heel gait still present	Mild dorsal bunion, 1st MP joint; moderate planus	Night brace to keep tendo achillis stretched is worn
44. G. T. Left	Toe-heel gait (brace required); cavus	AT = 4 GS = 5 PT = 4 P = 3 EDL = 5 FDL = 4 EHL = 5 FHL = 4	12/31/56, transfer of EDL and EHL: 2 to medial cuneiform, and 3 to 3rd metatarsal	8	9	Good dorsiflexor actjvity; only slight limp; heel-toe gait	No cavus	None

(a)† Transfer of peroneus brevis and longus to "mid-tarsal region", 8/18/50. Power not adequate for dorsiflexion.
(b)†† Extensor transfer preceded by transfer of peroneus longus to calcaneus.
(c)††† Peroneus brevis only; peroneus longus had been transferred to lateral cuneiform without benefit.

of Friedreich's ataxia with cavus deformity, claw toes, and ineffective dorsiflexor power.

In the cases presented in Table I, the common aim and purpose of the toe-extensor transfers was improvement of dorsiflexor function of the foot and ankle. The side-to-side balance of the foot and ankle to some extent influenced the surgeon in selection of the site of attachment of the toe-extensor transfers. Varus deformity of the foot and ankle was an indication for more lateral placement of the toe-extensor transfers. In eight of the forty-four cases presented in Table I (Cases 1, 8, 15, 35, 38, 39, 40, and 44), the muscle power of the anterior tibial was rated as fair or better, and in most of these cases we observe the paradoxical

FIG. 1-A

Fig. 1-A: Case 24. August 10, 1951. Residual absence of the anterior and posterior tibial muscles due to onset of poliomyelitis one year previously, causing drop-foot, valgus deformity, and pes planus on the left.

Fig. 1-B: Left foot, September 7, 1956, immediately prior to toe-extensor transfer. Attempted dorsiflexion of the foot causes marked toe-extensor substitution.

Fig. 1-C: September 7, 1956. Position of the foot and ankle immediately following attachment of five long toe extensors to the medial cuneiform and tendo achillis lengthening.

Fig. 1-D: January 9, 1958. Active dorsiflexion of the left foot from the toe-extensor transfers. Secondary procedure was done on August 26, 1957, transferring the flexor digitorum longus and flexor hallucis longus to the undersurface of the first metatarsal to replace the posterior tibial and to prevent pes planus.

Fig. 1-E: January 9, 1958. On weight-bearing both feet have slight pes planus.

FIG. 1-B

FIG. 1-C

situation of ineffective dorsiflexor activity in a foot and ankle in which the anterior tibial, by separate muscle grading, shows satisfactory strength, most frequently noted in feet with some tendency to cavus deformity. Hibbs hinted at this situation when he described a dropping down of the front of the foot in claw-foot and recommended toe-extensor transfer for correction of this dropping. I am consid-

erably hampered, however, in giving any further interpretation of Hibbs' article, because he made no attempt to assign an etiology or to describe the muscle power in the series of claw-foot which he presented. The presence of ineffective dorsiflexion, in spite of good muscle power in the anterior tibial, might indicate a need for transfer of the anterior tibial from its ineffective attachment to a more effective placement on the dorsum of the medial cuneiform in addition to the toe-extensor transfers; anterior tibial transfer was not used, however, in the cases described in Table I.

In this series, transfer of the five long toe-extensor tendons was used in preference to transfer of the peroneus longus for replacement of the paralyzed anterior tibial because of the following advantages:

FIG. 1-D

FIG. 1-E

1. When they are transferred together, the five long toe extensors are believed to have more power than a single peroneal;

2. The toe-extensor tendons are retained in their own sheath, and little effort is required in moving the tendons to their new insertions;

3. The long toe extensors are already auxiliary dorsiflexors of the foot and are easily trained to dorsiflex the foot and ankle after they have been transferred. This finding is in accord with the electromyographic studies of Close [2,3], which showed that toe extensors have the same phasic activity as the anterior tibial which is being replaced.

A frequent finding in a foot with a paralyzed anterior tibial is marked toe-extensor substitution on attempted dorsiflexion of the foot and ankle (Fig. 1-B), indicating that the toe extensors can easily be trained to dorsiflex the foot and ankle if properly attached.

AGE LIMITS

It is usually desirable to wait until the child is old enough to cooperate in an adequate muscle test, which he should be able to do at the age of four or five years. An equinus contracture occurring in a younger child should be controlled by a release of the tight Achilles tendon and, if needed, by a posterior capsulotomy of the ankle; the foot should be supported in a satisfactory position in a brace. Tendon transfers are then deferred until the child is old enough to cooperate in a satisfactory muscle test. There is no upper age limit, but the development of fixed deformities in older persons who have had poliomyelitis during their childhood may make tarsal fusion necessary. In such cases toe-extensor transfers may still be a good source of dorsiflexor power and balance the foot when used in conjunction with a triple arthrodesis.

GUIDES TO PROPER PLACEMENT OF THE TENDONS

Figure 2, which is an anteroposterior roentgenogram of a foot and ankle in extreme equinus position, shows that a line extended perpendicularly from the mid-point of the talocrural joint runs through the second ray. This indicates that the second toe and metatarsal are the mid-line of the foot. In actual practice it has been noted that even a tendon placed at the base of the second metatarsal will act as a mild evertor, and as tendons are placed farther laterally from this point, progressively more active eversion is obtained from the transfer. If the posterior tibial is completely absent, placement of all five tendons in the medial cuneiform is indicated; nevertheless, the foot will still drift into valgus deformity if a weak tibialis posterior is not replaced by an appropriate transfer.

FIG. 2

Anteroposterior roentgenogram of foot and ankle with the foot in marked equinus deformity. The mid-line of the foot is shown.

The correct placement of a tendon transfer on the foot is demonstrated by Line A in Figure 3. If the tendon is attached too far distally (Line B), the lever arm is long, and excessive excursion of the muscle is required before adequate dorsiflexion of the foot is obtained. The direction of pull is further modified by the change in direction of the tendon as it extends subcutaneously out onto the distal portion of the foot to its insertion (Line C)*. A tendon inserted too far proximally in the navicular or neck of the talus (Line D) will require much less excursion for adequate dorsiflexion of the foot but will require excessive power from the transferred muscle because of the short lever arm. A good compromise between excursion and power, therefore, can be obtained by attachment of the tendon transfers in a zone which includes the medial cuneiform, the bases of the second, third, and fourth metatarsals, and the cuboid (Fig. 4). Attachment to the bases of the first and fifth metatarsals is usually avoided because the resulting dorsal displacement of the first and fifth metatarsal heads is generally undesirable in a major weight-bearing bone and frequently results in an annoying dorsal bunion. Placement of the tendon into the base of the first metatarsal is also hampered by the presence of an epiphysis in this area but may occasionally be indicated if the surgeon wishes some dorsal displacement of the first metatarsal to correct cavus deformity. Green and Grice stated that dorsal bunion of the first metatarsophalangeal joint may be caused by severance of the peroneus longus tendon, which releases the downward pull on the first metatarsal. However, dorsal bunion can also occur without previous severance of the peroneus longus tendon. When this occurs after toe-extensor transfers, it is believed to be due to dorsal pull on the first metatarsal and medial cuneiform (Fig. 5).

METHODS OF TENDON ATTACHMENT

Tunnels are drilled in either the medial cuneiform or bases of the second,

* An example of this type of attachment is the Jones transfer of the extensor hallucis longus to the first metatarsal neck.[7] This procedure was originally described for treatment of cock-up deformity of the great toe and was not used to supply dorsiflexor power to the foot and ankle.

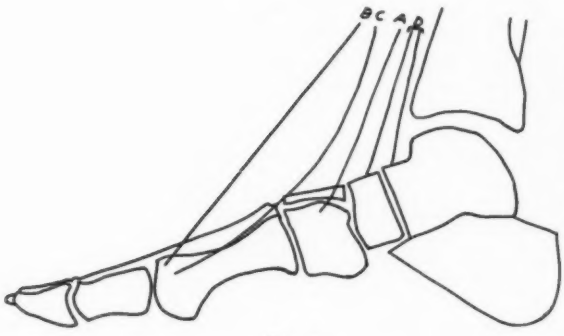

FIG. 3

Fig. 3: Correct placement of the tendon insertion on the foot-ankle lever arm (line *A*).

Fig. 4: Recommended sites of tendon attachment. Only rarely in post-poliomyelitic paralysis is it necessary to place the tendons as far laterally as the fourth metatarsal base or cuboid.

Fig. 5: Dorsal pull on the base of a metatarsal causes dorsal displacement of the metatarsal head.

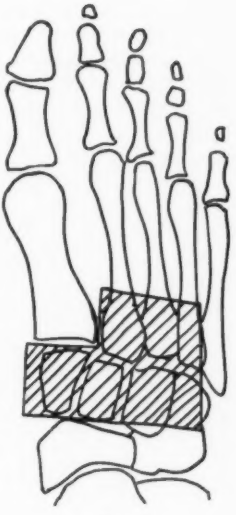

FIG. 4

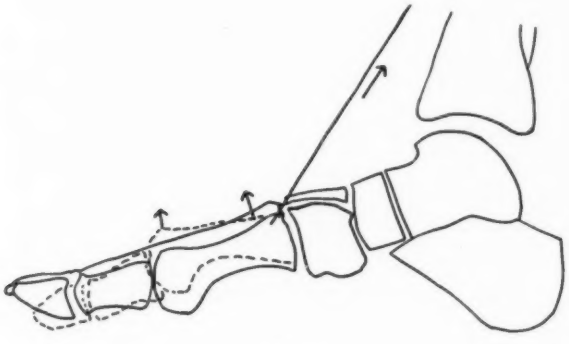

FIG. 5

third, or fourth metatarsals. If more lateral attachment is desired in order to obtain more evertor strength, attachment to the cuboid may be considered, but cuboid attachment has rarely been used at this hospital for foot imbalance due to poliomyelitis. Cuboid attachment has been used in selected cases of resistant club-foot in which more evertor and dorsiflexor pull is desired. The tendon, or tendons, being transferred are pulled through these tunnels and sutured to themselves under adequate tension. The Bunnell pull-out wire is not used because, with many tendons, if put under adequate tension, undue skin pressure may result from the button or bolus used to secure the pull-out wire and inadequate tension may result. With the tendons passed through a tunnel in the bone, doubled back and sutured to themselves, the desired amount of tension is easily obtained and controlled (Figs. 6 and 7).

TECHNIQUE OF OPERATION

Prior to scheduling of the procedure, a careful muscle examination of the foot

and ankle is performed by the surgeon in order to determine the proper site or sites for attachment of the transfers. This task should not be relegated to the physical therapist because the surgeon can gain considerable information about the balance of the foot and ankle, and about the indicated sites for tendon attachment, by performing the muscle examination himself. For isolated anterior tibial paralysis all five tendons are usually listed for insertion into the medial cuneiform. If there is some tendency to inversion of the foot due to peroneal weakness,

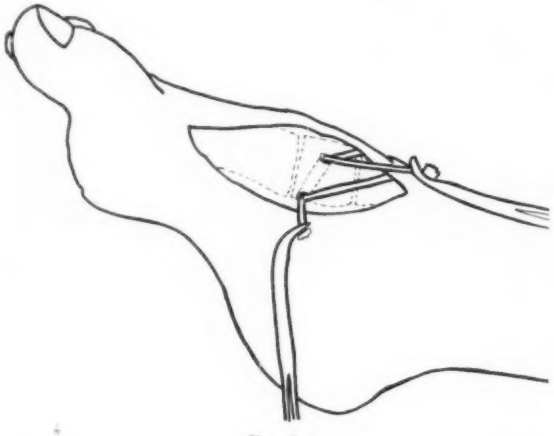

FIG. 6

Attachment of long toe-extensor tendons to the medial cuneiform.

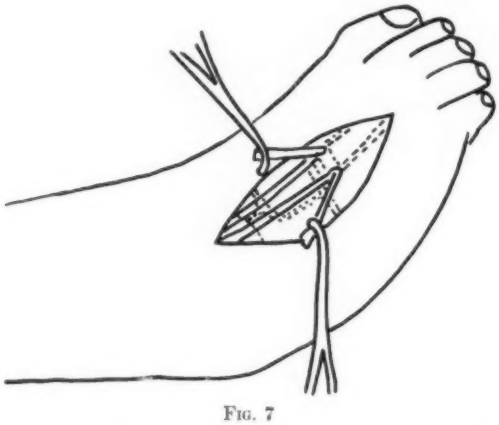

FIG. 7

Attachment of long toe-extensor tendons to a metatarsal base.

two or three tendons may be diverted for transfer into the second or third metatarsal base. The temptation to place the tendon attachments too far laterally should be diligently avoided because a residual valgus position of the foot is a frequent result. Only with a severe varus deformity of the foot is it desirable to attach the tendons to the fourth metatarsal base or cuboid. In any event, one or two tendons should always be attached to the medial cuneiform to support the medial ray (first metatarsal and medial cuneiform) to prevent dropping of the first meta-

tarsal. Observation at surgery has shown that dorsal pull on the medial cuneiform, first metatarsal base, or any part of the first metatarsal, will cause dorsal displacement of the metatarsal head (Fig. 5). Attachment of the tendons to these bones will, therefore, tend to decrease or prevent cavus deformity and if unopposed may cause a marked dorsal bunion.

FIG. 8

Case 39. Fracture of the fourth metatarsal at the site of tendon attachment. Roentgenogram was made about fifteen months after tendon transfer. The dorsiflexor transfer was still active in spite of fracture at the site of tendon attachment.

With adequate sterile precautions and under tourniquet control, longitudinal incisions are made on the dorsum of the foot medially over the first metatarsal and medial cuneiform and laterally between the third and fourth metatarsals extending proximally between the lateral cuneiform and cuboid. The incisions should be far enough apart to preserve adequate blood supply to the strip of skin between the incisions in order to prevent skin slough (this precaution would be especially pertinent for surgery on an adult's foot). The extensor hallucis longus and extensor digitorum longus tendons are cut as far distally as possible from these incisions. Plantar-flexion deformity of the great toe is prevented by either fusion of the interphalangeal joint or attachment of the extensor hallucis brevis into the distal stump of the extensor hallucis longus. The tendons of the extensor digitorum longus to be attached to the medial cuneiform are passed subcutaneously to the medial wound and a dorsal-plantar tunnel is drilled through the medial cuneiform. The ends of the tunnel are widened with a curette and the tendons are pulled through either end of the tunnel with a wire loop, pulled to the desired tension

(Fig. 6), and sutured in place. Those tendons of the extensor digitorum longus which have been left in the lateral wound to be inserted into the base of the metatarsal are pulled through a transverse tunnel in the base of the metatarsal chosen for attachment (Fig. 7).

If the peroneus longus is to be used to reinforce the toe-extensor transfer for dorsiflexor power, it is cut distally as it passes under the lateral border of the foot, pulled into a wound on the lateral aspect of the leg, and directed through the anterior tibial sheath to the medial cuneiform where it can be sutured to the stumps of the transferred extensor tendons in the medial cuneiform. Since the article of Fried and Hendel was published, I have found that it is preferable to use the peroneus longus to replace the posterior tibial; the peroneus longus, therefore, is not considered available as a dorsiflexor transfer in the presence of weakness of the posterior tibial. Green and Grice caution that the distal stump of the peroneus longus should be sutured to the detached peroneus brevis to maintain downward pull on the first metatarsal. This extra step is now a part of the procedure but it was not done in the patients presented here. It is used only when the peroneus longus is being transferred for dorsiflexor function but is not used in the Fried and Hendel transfer in which the peroneus longus is being transferred through the posterior tibial sheath to replace the tibialis posterior.

If the Achilles tendon is contracted, or if there is any chance that the gastrocnemius-soleus group may overpower the dorsiflexor transfer, the Achilles tendon must be lengthened. A posterior capsulotomy of the ankle joint should be performed if adequate dorsiflexion still cannot be obtained. There is, however, one exception: tendo achillis lengthening should be delayed, as recommended by Hibbs, if toe-extensor transfer is done and correction of a cavus deformity is desired.

The skin incisions are closed with No. 30 subcuticular running wire sutures. This type of suture is non-irritating and may be left in the skin without the necessity of cast changes before the final cast removal. A leg cast is applied with the foot dorsiflexed at least to neutral.

The cast is removed six weeks after surgery, the wire skin sutures are removed, and the patient is instructed in active dorsiflexion of the foot. It is usually sufficient to instruct the patient to think of lifting his toes when he lifts his foot for the swing phase of his gait.

RESULTS

In Table I are summarized the early follow-up findings of forty-four consecutive long toe-extensor transfers performed for postpoliomyelitic muscle imbalance about the foot and ankle, covering a period from May 1956 through December 1956. Thirty-eight of the forty-four patients were followed for more than fifteen months; the follow-up period for the whole group varied from five to twenty-three months, with the exception of one patient (Case 26) who had only a single follow-up examination at three months and did not return for further examination and services. Of the forty-four patients, twenty-nine had good or better active dorsiflexion of the foot, five had fair active dorsiflexion, and ten had poor or no active dorsiflexion. Of the ten patients with poor or no active dorsiflexion, four had to be refitted with drop-foot braces, and when one of these four reached the age for arthrodesis a pantalar arthrodesis was performed. Two of the ten patients required little or no further treatment—one got along nicely with a high-top shoe and the other had a tenodesis effect from the transfer, limiting plantarflexion range. Four of the ten operations that were failures resulted in improvement when further tendon transfers were performed to reinforce the toe-extensor transfers.

Six of the entire series of forty-four patients had residual equinus contractures with limited dorsiflexor range. Two of the six responded to tendo achillis lengthening and further tendon transfers, one responded to stretching exercises, and two responded to support of the foot with a drop-foot night brace. In the one re-

maining case of equinus contracture reinstitution of permanent bracing was required.

The extensor digitorum longus was transferred alone in only two patients (Cases 5 and 9). Both of these patients required further tendon transfers to obtain satisfactory dorsiflexor power and, therefore, transfer of the extensor digitorum longus alone, although an easier operative procedure, appears to be much less successful than transfer of both the extensor digitorum longus and extensor hallucis longus.

The most striking residual deformity in this series was either pes planus or valgus deformity of the foot, or both, which occurred in varying degrees in twenty of the forty-four patients. The severity of this deformity seemed to be related to the amount of weakness in the tibialis posterior muscle. In eight of these, transfer of the peroneus longus through the posterior tibial sheath or transfer of the long toe flexors to replace posterior tibial strength was performed as a secondary procedure. In two additional patients a similar transfer to replace posterior tibial weakness was combined with a modified Durham flat-foot procedure [1]. The number of these secondary procedures is too small to permit any vital conclusions as to the results, however, it is worth noting that in the two patients in whom transfer of the peroneus longus through the posterior tibial sheath (Fried and Hendel procedure) was performed, the valgus deformity and pes planus were suitably corrected. Moderate improvement of valgus deformity and pes planus was also obtained by transfer of the long toe flexors to replace the tibialis posterior in five patients.

COMPLICATIONS

Theoretically, an expected complication might be imbalance or deformity of the toes owing to release of the long toe extensors, but although this complication was diligently watched for, it did not occur in this series to an extent significant enough to be picked up on examination. Actually, it was noted that cock-up deformity of the toes in some cases, which was present before operation owing to toe-extensor substitution, was considerably improved or corrected by the toe-extensor transfers.

There were no postoperative infections in this series. I believe that this was due to our ability to effect a thorough cleansing of the foot and ankle in every patient forty-eight and twenty-four hours prior to surgery.

Dorsal bunion occurred at the first metatarsophalangeal joint in only one patient, and I am again reminded of the advice of Green and Grice about proper attachment of the distal stump of the peroneus longus to the peroneus brevis when the peroneus longus is used as a dorsiflexor transfer. In three patients in whom the extensor digitorum longus was attached to the base of the fourth metatarsal there was a rather striking dorsal bunion of the fourth metatarsophalangeal joint, which in all cases was asymptomatic and did not bother the patient but was notable enough to be mentioned in the clinic notes each time the patient was examined. For this reason and other reasons to be mentioned later, it is recommended that no more than three tendons be attached to the base of any metatarsal.

In one patient (Case 39) late fracture occurred in a metatarsal at the site of attachment of four extensor digitorum longus tendons. The patient complained of rapidly developing pain in the right foot about fourteen months after toe-extensor transfer. A roentgenogram made on February 18, 1958, showed a non-united fracture of the fourth metatarsal (Fig. 8). The tendons evidently remained attached to scar tissue because the patient continued to have good active dorsiflexion of the foot and activity of the tendon transfers. On February 26, 1958, bone-grafting to the fourth metatarsal bone was performed, and the attachment of the extensor digitorum longus was moved to the lateral cuneiform. It is believed that this complication can be prevented if no more than three tendons are attached to any metatarsal base and if the tendons are attached as far proximally as possible

on the metatarsal into cancellous bone rather than to the slender cortical bone father, distally, on the metatarsal shaft.

DISCUSSION

It must be emphasized that the follow-up of the patients presented in Table I is in all instances less than two years. Further surgery will undoubtedly be required in some of these patients to provide balance or stability of the foot and ankle or to correct deformities. Although the follow-up findings in this short period are very valuable, and are probably more detailed than could be obtained after a longer follow-up period, it is almost certain that a longer period of observation could reveal changes in the balance of the foot with growth, which cannot be predicted on the basis of the findings presented here.

Two causes of complete failure—inadequate dorsiflexor activity after transfer—are noted in this series: (1) inadequate muscle power (fair or less), before operation, of the transferred toe extensors; and (2) excessive power or fixed contracture of the plantar flexors of the foot and ankle working against the dorsiflexor transfer. The importance of releasing equinus contractures and weakening the gastrocnemius-soleus pull by tendo achillis lengthening cannot be overemphasized. However, if correction of a cavus deformity by tendon transfer is desired, tendo achillis lengthening should be deferred. In an occasional patient with poor dorsiflexor power after toe-extensor transfer owing to overpowering of the transfer by a tight Achilles tendon, lengthening of this tendon will result in considerable improvement. One patient (Case 17) had poor dorsiflexor activity after a toe-extensor transfer on July 13, 1956, and again after the dorsiflexor transfer had been reinforced by transfer of the peroneus longus and tertius muscles on June 17, 1957. Dorsiflexor activity improved considerably after tendo achillis lengthening done on March 3, 1958, after which the patient wore a leg-holding cast for six weeks. Needless to say it is preferable to lengthen the Achilles tendon and to release the equinus contracture at the time of the original transfer if there is any chance that the dorsiflexor transfer may be overpowered by excessive plantar flexor strength or contracture.

It is to be expected that some of the patients in this series may require triple arthrodesis at a later age, especially those with side-to-side instability of the foot. This, however, is not considered to be a contra-indication to extensor transfer since an already existing active dorsiflexor transfer and the resultant improved form and position of the foot will enhance the final result considerably.

Although the basic principles outlined in this paper may be of value to the surgeon desiring to balance the foot and ankle by tendon transfer, the best method for balancing a foot with combined weakness or absence of the anterior and posterior tibial muscles is still uncertain. A review of Table I shows an undesirably large number of secondary tendon transfers required after toe-extensor transfers in those patients with weak or absent anterior and posterior tibial muscles. The experiences of Fried and Hendel had not been published when initial surgery was performed on the patients presented in Table I. In the light of this work it would now seem desirable to attempt to reduce the number of operative procedures on any given patient by combining the tendon transfer for dorsiflexor power with the tendon transfer for invertor power. I have done this more recently, but it is too early to report the results on this single-stage combined procedure. A more conservative approach would be a planned two-stage procedure, possibly transferring the peroneus longus through the posterior tibial sheath for invertor strength as recommended by Fried and Hendel, followed in a few months by a careful muscle test and evaluation of the balance of the foot to guide the surgeon in placement of the toe-extensor tendons to provide dorsiflexor power at the second stage.

SUMMARY AND CONCLUSIONS

1. A method for correcting imbalance and improving dorsiflexor activity of

a paralyzed foot resulting from poliomyelitis by means of combined extensor digitorum longus and extensor hallucis longus transfers has been described and some short-range follow-up observations outlined.

2. When present, posterior tibial weakness must also be replaced by means of tendon transfer if satisfactory balance of the foot is to be obtained without tarsal fusion.

3. With the exception of the cavus foot requiring correction, equinus contractures should be overcome, preferably by tendo achillis lengthening at the time of tendon transfer and, if necessary, posterior ankle capsulotomy prior to the toe-extensor transfer.

4. The use of long toe-extensor transfers for dorsiflexor power, if combined with the method of Fried and Hendel, when indicated for the replacement of posterior tibial weakness, should allow the surgeon to obtain balance of the paralyzed foot at an early age, allowing certain patients to discard braces early without the necessity of tarsal fusion.

5. The exact order of the procedures to improve dorsiflexor power and to improve invertor power has yet to be determined. Three possibilities exist: (1) a two-stage procedure with tendon transfers to improve invertor power as recommended by Fried and Hendel, followed in a few months by toe-extensor transfers to improve dorsiflexor power; (2) a two-stage procedure with initial toe-extensor transfers for dorsiflexor power followed in a few months by the tendon transfer described by Fried and Hendel and used in some cases in this series to improve invertor power; (3) a single-stage procedure combining the tendon transfers for dorsiflexor power with the Fried and Hendel tendon transfers for invertor power. The first two-stage procedure listed is probably the most conservative and the most accurate because it offers to the surgeon a chance for evaluation by a muscle test before final placement of the dorsiflexor transfers.

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Alterations in Growth-Cartilage in Experimental Dwarfism*†

I. Studies on Insulin-Dwarfed Chicks

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Reports of the production of dwarfism and skeletal deformities in chick embryos are well known. Insulin, sulfonamides, adrenal steroids, heavy metals, benzyl alcohol, various enzyme inhibitors, and thallous salts have all been used to produce skeletal abnormalities.

The exact mechanisms by which these agents produce alterations in growth have received little experimental study and are poorly understood.

Duraiswami, Zwilling, Landauer, and Zambotti implied that alterations in the metabolism of carbohydrates of the developing embryo are responsible for the deformities produced by some of these agents. Duraiswami stated that glycogen and acid mucopolysaccharides (the major carbohydrate components of cartilage) are involved in the insulin-induced disturbances in the chick embryo. Zwilling demonstrated hypoglycemia lasting as long as seventy-two hours in the embryonic circulation of the chick after a single injection of insulin into the yolk sac. He noted also that in those embryos with the most severe hypoglycemia, the most severe dwarfism and deformities developed. Nicotinamide and riboflavin exert a protective action against the dwarfing effect of insulin and the sulfonamides. Follis, Rubin and Howard, Gutman and Yü, and Albaum and associates also presented evidence which associated glycolysis directly with the growth and calcification of cartilage.

Assuming that disturbance in carbohydrate metabolism is responsible for the development of dwarfism and skeletal deformity after the injection of insulin in chick embryos, one would expect some demonstrable alteration in the content, distribution, or character of the major carbohydrate components of the skeletal cartilage of dwarfed embryos.

This is a preliminary report on some of the studies we have undertaken to test this hypothesis.

METHODS AND MATERIALS

Eggs of the Hy-Line strain of white leghorn chickens were used. This strain is known to be free of spontaneously occurring skeletal deformities. The yolk sacs of 369 eggs were injected with from three to six units of crystalline insulin in 0.05 milliliter of solution, at periods ranging from seventy-two to 120 hours of incubation. Most of the injections were of six units of insulin at ninety-six hours. Controls were injected with 0.05 milliliter of physiological saline solution. Another group of 120 eggs was incubated without injection. All eggs were opened at eighteen days of incubation. The chicks were detached from the yolk sac and membranes, blotted dry, weighed, and their characteristics noted. Seventy-nine of the insulin group (31 per cent of the survivors) showed marked inhibition of skeletal growth, or serious skeletal deformity. Representative specimens of the deformed group were selected for further study, together with controls chosen at random.

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TABLE I

DWARFISM AND DEFORMITIES AFTER YOLK-SAC INJECTION OF INSULIN IN 72 TO 120-HOUR CHICK EMBRYOS

	Insulin Injected (*U-3 to U-6*)	Controls	
		Saline Injected	Non-injected
Number of eggs	369	63	120
Dead before eighteen days	94	3	6
Survivors	275	60	114
Dwarfed or deformed	79	2	2
Percentage "abnormal"	31	3.2	1.8
Type of Abnormality			
Beak deformity	15	0	0
Shortening of limbs	34	0	0
Kyphoscoliotic tibiae	21	0	0
Foot deformity	37	0	0
Feather deficiency	3	0	0
Spine deformity	1	0	0
Rumplessness	2	0	0
Other	3	0	0
Total	116	0	0
Dwarfism without other defect (weight less than 18 grams)	17	2	2

TABLE II

GLYCOGEN CONTENT OF SKELETAL CARTILAGE OF NORMAL AND INSULIN-DWARFED CHICK EMBRYOS*

	Control	Dwarf
Average of individual samples	1.31±0.34	1.34±0.41
Pooled samples		
10 saline controls	0.84	
10 normals	0.76	
9 dwarfs		0.91

*All values are shown as milligrams of glycogen per gram of wet weight of cartilage.

The skeletal cartilage about each knee joint was dissected free of soft tissue and removed by transecting the tibia and the femur in their middle thirds.

Fixation was carried out in Bouin's solution, chilled acetone, or neutral formalin. Rapid freezing and immediate frozen sections were used for determination of phosphorylase and glycogen. Freeze-substitution with acetone or absolute alcohol, or rapid freeze-drying was used for alkaline phosphatase. When required, decalcification was carried out using versene (ethylene-diamine-tetra-acetic acid) in saturated solution at room temperature. With the exception of the frozen sections, all tissue was sectioned at six to eight micra. Staining techniques used were: thionin and toluidin blue for metachromasia; Hotchkiss' modification of periodic-acid Schiff and Best's carmine for glycogen; and Gomori's aldehyde-fuchsin. Phosphorylase was determined by the method of Takeuchi and alkaline phosphatase by Gomori's technique. The von Kossa stain was used for calcified tissue.

For determination of their glycogen content, the skeletal cartilages about the knee joints were cleaned under a dissecting microscope. All calcified and connective tissue was cut away. The remaining cartilages were weighed, finely ground with sharp sand, extracted with normal saline solution, and the fluid extract analyzed for glycogen content. Individual knee-joint areas and pooled samples were analyzed by the method of Kahan.

Another group of forty eggs was injected with insulin at ninety-six hours, and with 350 microcuries of carrier-free S^{35}, as sodium sulphate, at seventeen days. An equal number of controls were injected with saline solution at ninety-six hours, and with 350 microcuries of S^{35} sulphate at seventeen days. Twenty-four hours later, the eggs were opened. One knee of each embryo was used for histological study and autoradiography. The opposite knee was dissected free of calcified and connective tissue, weighed, dissolved in hyamine [9], suitably diluted, and the radioactivity determined in a liquid scintillation counter. Results were interpreted as counts per milligram of cartilage tissue.

Cell and Matrix-Volume Studies

Early in this work we were surprised by the apparent marked cellularity of the hypertrophic cartilage area of the dwarfed specimens and the apparent paucity of cartilage matrix in this zone. This suggested a disproportion in the ratio of matrix to cells in the dwarfs, and we sought to measure this by microscopic methods.

Suitably stained sections of the extremities from thirty insulin-dwarfed chicks and thirty control specimens were microphotographed at a fixed magnification of approximately 400. The number of cartilage cells in each field of hypertrophic cartilage was counted and the average diameter of the cell was determined by measuring several samples of 500 cells in each group. These measurements were made directly on the photographs and checked by direct microscopic observation using an eyepiece micrometer. To avoid errors of observation, some of the measurements and counts were carried out independently by two observers, with almost identical results.

Assuming that the cartilage capsules in the hypertrophic zone are round, the area occupied by the content of each capsule may be determined by applying the formula: area $= \pi r^2$.

The volume is obtained by using the factor for the thickness of the section. By calculation, it is possible to determine the average area or volume occupied by the cells and the area or volume occupied by matrix in each selected field.

RESULTS

General Observations

Of 369 embryos injected with insulin, ninety-four died before the eighteenth day of incubation. Of the 275 survivors, seventy-nine (31 per cent) showed marked generalized dwarfism or severe skeletal deformities. The incidence and type of deformities are listed in Table I.

Histochemical Findings

No marked or consistent differences were noted between the insulin-deformed and control specimens with regard to the staining reactions of metachromasia, periodic-acid Schiff treatment, glycogen staining, aldehyde-fuchsin staining, alkaline phosphatase, and phosphorylase reactions. Regional variations were encountered with the various staining techniques in individual specimens in both groups. Somewhat greater variability was noted in the sections from deformed specimens, especially in regard to the uniformity of staining with aldehyde-fuchsin and the reaction for alkaline phosphatase.

Calcification

Calcified cartilage is present as a sleeve or cylinder lying adjacent to, and under, the subperiosteal diaphyseal bone of the tibia and femur. Under ordinary circumstances, the long bone of the chicken contains no bony metaphysis, and no epiphyseal plate as is seen in mammals. In some of the more severely dwarfed specimens, and those with kyphoscoliotic tibiae, the entire central portion of the shaft is filled with interlacing trabeculae of well formed cancellous bone. Cartilage

islands are found in this zone, and this cartilage is calcified. The pattern of calcification of cartilage is otherwise identical in the dwarfed and in the control groups.

Glycogen Content of Skeletal Cartilage

The cartilage in the knee area of ten control specimens was analyzed chem-

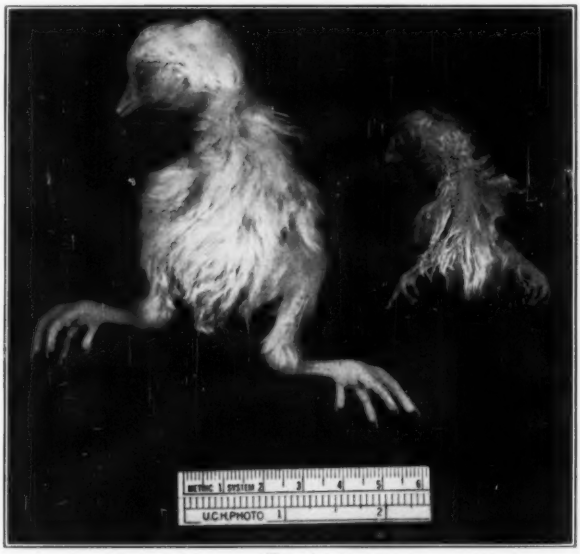

FIG. 1

Dwarfism and shortening of the extremities in a chick after yolk-sac injection of six units of insulin at ninety-six hours. On the left is the normal control at eighteen days and on the right is the insulin-dwarfed chick at eighteen days.

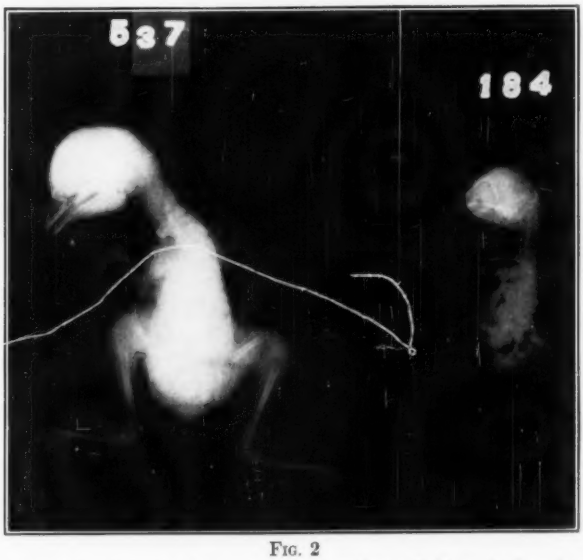

FIG. 2

Roentgenograms of a normal chick embryo (left) and of an insulin-dwarfed eighteen-day chick embryo (right). The dwarfed embryo shows shortening and bending of the tibia.

TABLE III

SIZE RELATIONSHIPS OF CELL TO MATRIX IN HYPERTROPHIC CARTILAGE ZONES OF INSULIN-DWARFED CHICKS

	Control	Dwarfed
Average number of cells per high-power dry field	55.2±6.2	73.2±7
Average cell diameter	28.6μ	26.36μ
Average individual cell area	642μ²	545μ²
Average total cell area per high-power dry field	35,444μ²	40,023μ²
Total field area	81,553μ²	81,553μ²
Average matrix area per high-power dry field	46,109μ²	41,530μ²
Average matrix area per cell	836μ²	567μ²

ically for glycogen content; the values ranged from 0.62 to 1.9 milligrams of glycogen per gram of tissue, with an average of 1.31 ± 0.34. In fifteen knees of dwarfed specimens the values ranged from 0.64 to 2.8, with an average of 1.34 milligrams per gram of tissue, ±0.4. Pooled samples of ten saline-injected controls, and twelve non-injected controls gave values of 0.84 and 0.76 milligram per gram of tissue, whereas a pooled sample of nine dwarfed knees gave a value of 0.91 milligram per gram of wet cartilage.

FIG. 3-A

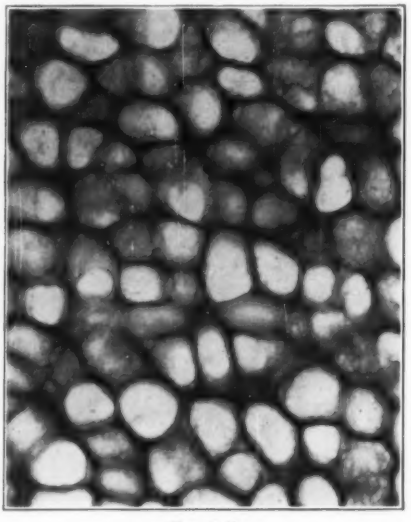

FIG. 3-B

Hypertrophic cartilage of chick embryo tibiae: control (Fig. 3-A and insulin dwarfed (Fig. 3-B).

Cell-to-Matrix Ratios

The average number of cartilage cells present per high-power field in thirty sections of control hypertrophic cartilage was found to be fifty-five. An equal number of fields in thirty dwarf sections gave an average of 73.2 cells per high dry field. This finding was at first interpreted as indicating that there was relatively less matrix and more cell volume in the hypertrophic zone of the dwarfs than in the normal controls.

The diameters of the cartilage capsules were then measured in the same fields;

groups of 500 capsule-diameter measurements were averaged, and from these findings the average area of the cartilage cells was determined. The findings are summarized in Table III. It was further observed that the greater the degree of dwarfing, the smaller and more numerous were the cells in the hypertrophic zone. Cell counts and measurements in resting and proliferating zones of cartilage revealed no significant difference in the number of cells per field or in the cell diameters in the dwarfed and control groups.

S^{35} Uptake Studies

The uptake by the embryo of S^{35} as sodium sulphate injected into the yolk sac varies greatly from one egg to another. The reasons for this are not known, but it is believed to be related to the fact that at seventeen days a large part of the remaining yolk is in solid form, and that this tends to retain the small quantity of fluid containing the isotope and acts as a physical barrier to absorption by the embryonic circulation.

Sixteen controls were injected with 350 microcuries of $S^{35}O_4$. Using the liquid scintillation counter, an average of 545 counts per milligram of tissue was obtained with a range of 127 to 1190 and a standard deviation of ±340. Ten dwarfed specimens gave an average of 555 counts per milligram, with a range of 244 to 1408 and a standard deviation of ±400.

DISCUSSION

It is to be emphasized that this is a preliminary report covering a number of pilot studies to discover further areas of investigation which might prove fruitful.

The use of chick embryos for studies of this kind has the advantage of ease of production of dwarfs, and a fairly large yield of material. Disadvantages of the chick-embryo method are the small quantity of tissue obtained from each egg, the need for a well controlled incubation system, and the rather wide divergence between the developmental pattern of a fowl and a human being. Despite these disadvantages, it is our feeling that the metabolic process, if considered on a tissue or cellular level, probably does not vary greatly from that of mammalian forms and that a great deal of valuable information may be obtained by the use of this method. So little of a factual nature is known about growth, metabolism, and cellular function of cartilage that this field is open to a wide variety of different approaches in an attempt to increase our knowledge of this tissue—so important to us in our work as orthopaedic surgeons.

Our findings suggest that in insulin dwarfism there is no marked change in the chemical composition of the cartilage cell or its matrix, at least in so far as the major carbohydrate constituents are concerned. Our findings suggest also that there is a constancy of the ratio—cell volume: matrix volume, and glycogen: total weight of cartilage—despite the severe deforming effects of insulin injected at an early period of development.

This would suggest that the injection of insulin does not alter the basic metabolic processes for utilization of carbohydrate, but rather deprives the cartilaginous tissue of those derivatives of carbohydrate from which the matrix may be fabricated.

In a study of this type, it would be desirable to be able to determine more accurately the quantity of chondroitin sulphate in each given area of cartilage matrix. Existing methods for separation of chondroitin sulphate from protein, and accurate quantitation of the small quantities of chondroitin sulphate thus liberated are not sufficiently standardized to permit accurate, reproducible results. Progress is being made in the development of such methods and it is hoped that future studies may be able to provide accurate quantitative measurements of matrical components from such small samples as those obtained in these experiments.

CONCLUSIONS

1. The hypertrophic cartilage zone of the lower extremities of insulin-dwarfed chicks contains significantly less matrix per individual cell than found in the same areas of normal controls.

2. The greater the degree of dwarfism, the smaller the cartilage cell, and the less matrix per cell is noted in the hypertrophic zone.

3. The ratio of total volume of cells to volume of matrix in hypertrophic cartilage is not significantly altered in insulin dwarfism in chick embryos.

4. Glycogen content, S^{35} uptake, and histochemical reactions for metachromasia, Schiff reaction, alkaline phosphatase, and phosphorylase do not appear to be significantly altered in the preosseous cartilage of the extremities in insulin-induced dwarfism in chick embryos.

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The Effect of Arteriovenous Fistulae on the Vascular Pattern of the Femora of Immature Dogs

A Microangiographic Study *†

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The paucity of knowledge on blood supply of bone is readily apparent to those interested in this fundamental factor of bone growth and bone metabolism. This fact has been emphasized by Trueta. Tilling recently reviewed the literature on the vascular anatomy of long bones, but only twenty-three articles appeared in his bibliography. This small number indirectly points up our limited knowledge of this subject.

The experimental work which forms the basis for this paper is part of a large project being carried out at the Mayo Clinic to investigate the blood supply of bone. The technique of microangiography being used in this study has been reported [10]. Other methods also being used at the Mayo Clinic have been described by Haliburton and co-workers in their report on the blood supply of the talus.

The fact that an arteriovenous fistula would increase bone length was noted in 1870 by Broca, and in 1950, one of us (J. M. J.) and Musgrove reported that arteriovenous fistulae would promote the growth of bone in immature dogs. Pearse and Morton, and Wu and Miltner also alluded to the effect of blood supply on bone growth. Their publications, and a translation of a monograph by Bier, provide an extensive bibliography on some of the early observations on the relationships between blood supply of bone and bone growth. Hutchison and Burdeaux have increased the length of bones in dogs by using tourniquets to obstruct venous return.

In the last two years, microangiography has appeared to be a valuable technique for our study of the effect of an arteriovenous fistula on bone, and we have carefully reviewed the development of this specialized technique of microangiography.

MATERIALS AND METHODS

During the first three months of 1957, twenty-six arteriovenous fistulae were constructed in mongrel puppies when they were estimated to be three and a half to four and a half months old. The fistulae were made at this age because work by Schlotthauer and Janes has indicated that the dogs' epiphyses remain open until the animals are eleven months old. Sixteen of the dogs were observed for periods of nine to fifteen months before being killed. Ten of the twenty-six dogs were omitted from the series because of death or obvious technical failure in construction of the fistulae as determined by auscultatory or palpatory findings; the criterion used was absence of a murmur and a thrill at the site of construction of the fistulae shortly after the surgical procedure. Of the sixteen dogs carried to the termination of the study, twelve had successful, well functioning fistulae. Two animals had closed fistulae at the time of the termination, and two had fistulae

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‡ The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

that were considered as functioning poorly. Of the twelve successful fistulae, eight were between the iliac artery and vein and four were between the femoral artery and vein.

One month following surgical formation of the fistulae and one month before the animal was killed, temperatures were recorded from the surface of the femur at the greater trochanter, at the middle of the shaft, and near the femoral condyle by means of thermistors mounted in needles. Since the temperature of the medullary portion of bone had been studied previously and a report had been made by one of us (J. M. J.) and Musgrove, these studies were not repeated.

The technique of preparing specimens for microangiographic study and the equipment needed were described in a previous publication [10]. A suspension of barium sulphate (micropaque) in gelatin was used in all cases as an injection mass. We routinely used physiological pressures for injection.

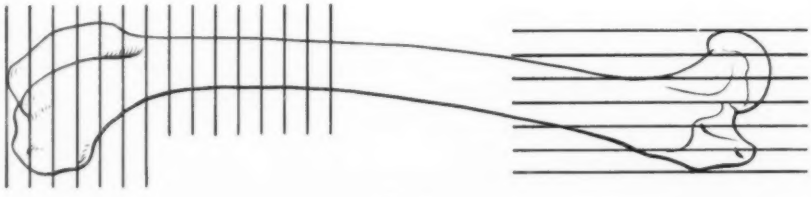

FIG. 1

Diagrammatic representation of the location of the sections of bone (one millimeter thick) that were prepared from the distal and proximal parts of the femur on the side of the fistula and also from the femur on the control side.

FIG. 2-A

FIG. 2-B

Figs. 2-A and 2-B: Gross arteriograms of the hind limbs of the dog at time of death.
Fig. 2-A: Marked vascularity is evident in the soft tissues on the side of the fistula on comparison with the control side.
Fig. 2-B: In this animal the fistula failed to function, and there is no apparent difference in vascularity of the soft tissues between the right and left hind limbs.

Extreme care, however, was taken in cannulating the aorta so that all terminal branches of the aorta were injected equally. Gross arteriograms of the abdomen and both hind quarters were obtained before removing the femora and tibiae. The gross arteriograms provided a check on our technique of injection and also permitted an estimation of vascularity of the soft tissues.

After the gross arteriograms were made, the fistulae were carefully dissected and the diameters of the artery and vein were measured. Then the femur and tibia

from both the side of the fistula and the side that had no fistula (the control side) were carefully removed and measured. Serial sections were obtained of the proximal and distal portions of the femur as previously reported by us. The industrial milling machine was found to be very good for making serial sections. The saw blades used were of the screw-head slotting variety and were 0.020 inch in thickness [8]. The method by which each femur was serially sectioned proximally and distally is shown in Figure 1. Sections were cut so that the proximal and distal epiphyses could be easily identified. Approximately 1,000 separate microangiograms were obtained in sixteen dogs.

RESULTS

Lengthening of the Bones (Femur and Tibia)

Increased length of the femur occurred in each hind limb in which a successful fistula was found. This is in agreement with previous findings reported by one of us (J. M. J.) and Musgrove. In the present series, the femur on the side of the fistula was lengthened in all twelve dogs with successful fistulae and the tibia on the same side was lengthened in nine of these twelve dogs. The femora were 2.7 millimeters longer on the average, with the range of one to five millimeters and the tibiae of nine animals had increased an average of 3.2 millimeters, with a range of 2.5 to four millimeters. It is worth noting that in all four dogs with fistulae between the femoral artery and vein, the length of the tibia had increased when compared with the normal side. The length was determined by direct measurement and checked by measurement on the roentgenogram. Roentgenograms were made of both femora and both tibiae at the same time.

Temperature Studies

The temperature on the surface of the femur in all twelve dogs with successful fistulae was found to be increased one month after surgical formation of the arteriovenous fistula and one month before the animals were killed. The average increase of temperature in the proximal part of the femur was 0.9 degree centigrade (range: 0.5 to 1.2 degrees), at the mid-femur, 1.0 degree centigrade (range: 0.5 to 1.8 degrees), and at the distal part 1.2 degrees centigrade (range: 0.7 to 2.0 degrees). In two limbs which had poorly functioning fistulae and in two in which the fistulae failed, the temperature of the femur was not elevated. Subsequent microangiographic studies revealed no alteration in the blood supply of the femora of these four dogs, and none of these four dogs had any lengthening of the femora or the tibiae.

Gross Arteriograms

Gross arteriograms, in addition to the macroangiograms, were made with the dogs in the supine position. These helped us in evaluating the accuracy of our technique of injection of both hind limbs. As near as we could tell, the inflow of micropaque suspension into both hind limbs was equal. We determined this by observing good filling of both tibial and femoral arteries, and also the branches from the aorta to the dog's tail. The macroangiograms also allowed observation of what most persons consider the collateral blood supply in the soft tissues of the thigh. Unquestionably some of the picture on the gross arteriogram or macroangiogram represented dilated veins, but we feel much of the collateral supply was arterial. Examples of our macroangiograms are shown in Figures 2-A and 2-B. Figure 2-A shows a successful fistula, and Figure 2-B shows the vessels in the limb in which the arteriovenous fistula had closed off about two months after the surgical formation.

Dissection of the Fistula

At the time that the animals were killed, the fistulae were carefully dissected, and measurements were made of the diameter of the artery, the diameter of the

FIG. 3-A

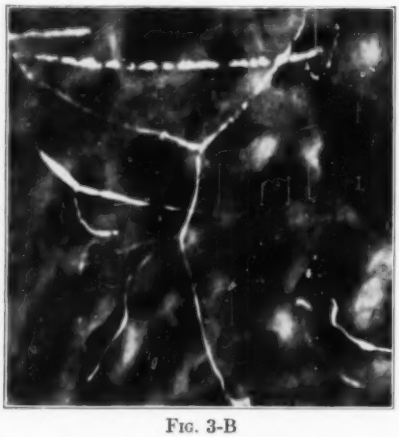

FIG. 3-B

Fig. 3-A: Microangiogram of a section (one millimeter thick) from the head of the femur near the epiphysis on the side of the arteriovenous fistula. Note the increased filling of vessels (× 30).

Fig. 3-B: Microangiogram of section of femoral head from control side in same dog (× 30). The section shown was in the same plane as the one shown in Fig. 3-A and was obtained from a comparable area of the femoral head near the epiphysis.

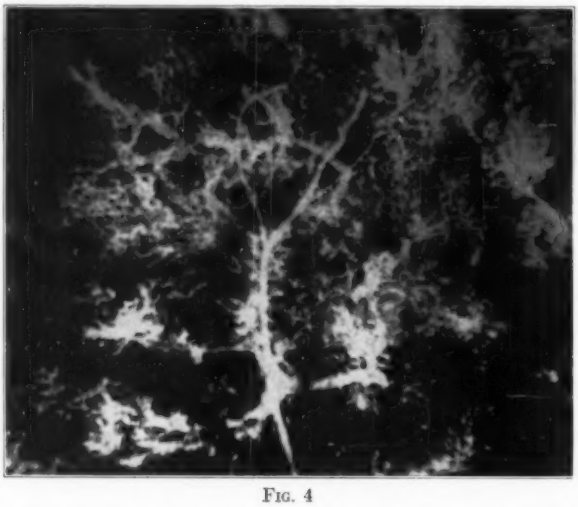

FIG. 4

Microangiogram of a section from the head of the femur on the side of the fistula. Vascularity is marked, and the capillaries and small vessels show marked arborization (× 30).

fistula, and the diameter of the vein. We found that the diameters of twelve of the fistulae were about equal to the diameter of the artery proximal to the fistula. Two fistulae which were less than one-half the diameter of the proximal artery failed to produce any vascular alteration in the bone and also failed to cause lengthening of the femur and elevation in temperature on the surface of the femur.

Microangiographic Findings

Consistent and constant changes were noted in the femora of twelve dogs on the side of the successful fistula. Briefly we noted the following:

1. Hypervascularity of the small vessels including capillaries of the medullary portion of the femur;

2. Increase in the cortical and periosteal blood supply, particularly in the posterior distal half of the femur and in the region of the distal femoral epiphysis;

3. In some animals a degree of dilatation of the individual vessels.

Hypervascularity: We use the term *hypervascularity* to indicate the presence of more filled vessels when specimens of femur from the fistula side are com-

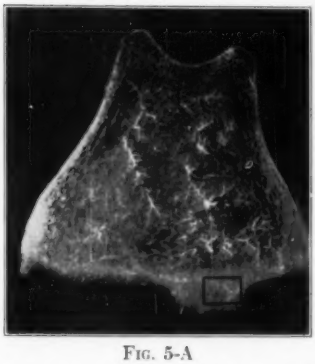

FIG. 5-A

FIG. 5-B

Figs. 5-A through 5-D: Microangiograms of sections from the region of the distal epiphysis of the femur of a dog.

Fig. 5-A: From the side of the arteriovenous fistula (× 1.2). Area outlined in black is shown in Fig. 5-B.

Fig. 5-B: Higher magnification of the portion of the posterior cortical and periosteal regions outlined in black in Fig. 5-A. There is marked increase in the vascularity of this area (× 30).

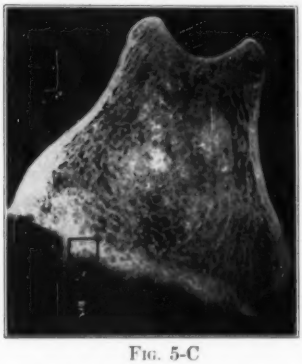

FIG. 5-C

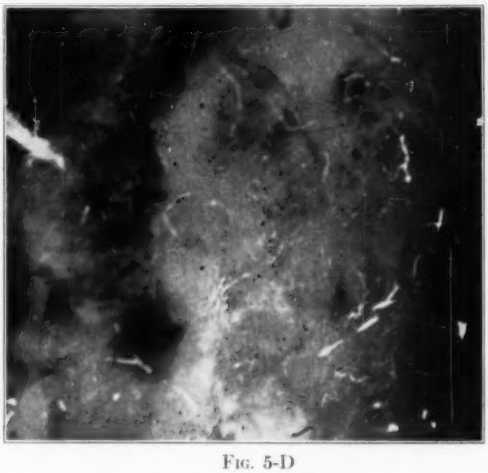

FIG. 5-D

Fig. 5-C: Identical section to that shown in Fig. 5-A, but from the control side (× 1.2).

Fig. 5-D: Higher magnification of the portion of the posterior cortical and periosteal regions outlined in Fig. 5-C. Fewer filled vessels are evident than in Fig. 5-B (× 30).

pared with similarly located specimens of femur from the other or control side. This was especially evident when vessels in the femoral head near the epiphysis on the side of the arteriovenous fistula were compared with those on the other (control) side (Figs. 3-A and 3-B). Besides hypervascularity, we observed a tendency to aborization of the small vessels. We use the word *arborization* to describe the vessel pattern that almost looks like a fir tree (Fig. 4).

Increase in the cortical and periosteal blood supply: Figures 5-A through 5 D show the tremendous increase in the number of filled vessels in the distal posterior part of the femur. The specimens show this change in the region of the distal femoral epiphysis and also proximal to that region.

Dilatation of the vessels: Figures 6-A through 6-D show quite definite dilatation of the vessels on the side of the fistula when compared with the other side. This dilatation of vessels was not a constant or consistent change. It was noted definitely in four of the dogs and seemed present in the others, but not as unequivocally. We doubt whether this dilatation represents an artefact as we obtained stereoscopic microangiograms of certain slices such as shown in Figure 7.

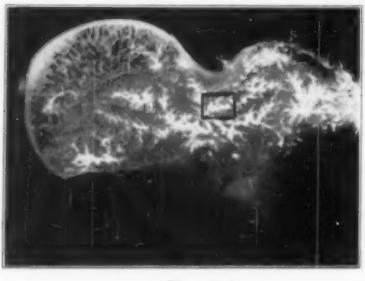

FIG. 6-A

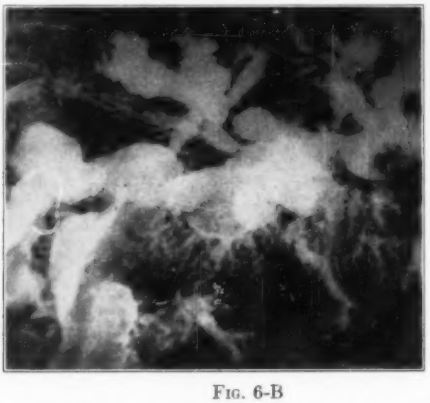

FIG. 6-B

Figs. 6-A through 6-D: Microangiograms of sections from the heads of both femora of a dog.

Fig. 6-A: From the side of the arteriovenous fistula (× 1.8). Area outlined in black is shown in Fig. 6-B.

Fig. 6-B: Higher magnification of portion of the medulla outlined in black in Fig. 6-A. The medullary vessels are markedly dilated (× 30).

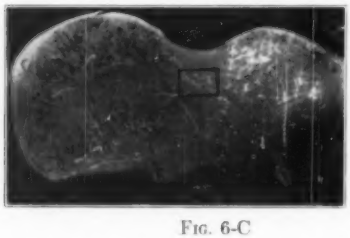

FIG. 6-C

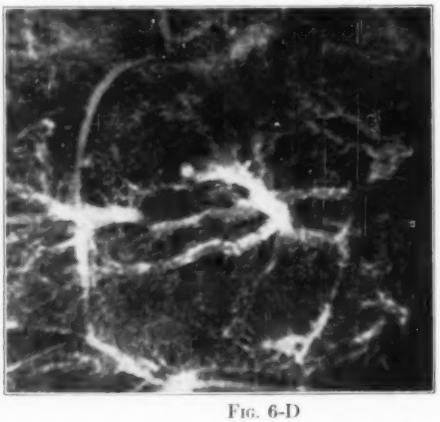

FIG. 6-D

Fig. 6-C: Identical section to Fig. 6-A but from the control side (× 1.65). Area outlined in black is shown in Fig. 6-A.

Fig. 6-D: Higher magnification of area outlined in black in Fig. 6-C. Dilatation of medullary vessels should be compared with that in Fig. 6-B (× 30).

COMMENT

We think with a fair degree of confidence that three definite changes take place in the limb of a dog with a well functioning fistula in the hind limb. They are:

1. Elevation of the temperature on the surface and in the intramedullary portion of bone (intramedullary bone temperature was previously reported by one of us, Janes, and Musgrove);

2. Lengthening of the femur;

3. A definite alteration in the blood supply of the femur as indicated by microangiograms.

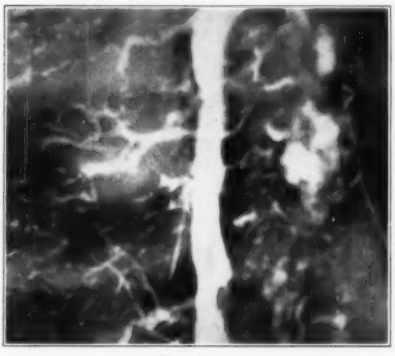

FIG. 7-A

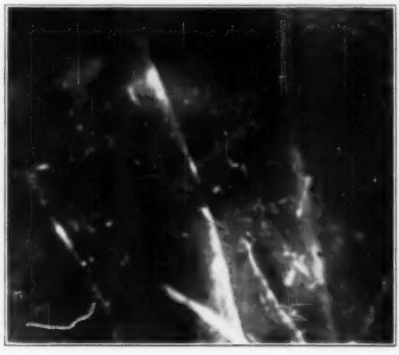

FIG. 7-B

Figs. 7-A and 7-B: Microangiograms of comparable sections of bone from the right and left distal femoral notches.

Fig. 7-A: From the side of the fistula. The dilatation of vessels and the increased vascularity on side of fistula should be noted (× 40).

Fig. 7-B: From the control side (× 40).

The findings from our microangiograms and our temperature studies indicate an increased blood supply in the femur at least. Robertson and associates and Henrie concluded from plethysmographic studies and use of radioactive albumin that blood flow is decreased distally in a limb with an arteriovenous fistula. With these writers we would agree, but as they pointed out, a tremendous collateral blood supply must develop about the fistula or the surgeon would never be able to excise an arteriovenous fistula. If, as Deterling said, the vessels seen in the gross arteriogram are all veins, then the excision of a fistula would nearly always lead to a gangrenous limb.

With the exception of Hutchison and Burdeaux, no one has demonstrated that pure venous stasis causes an increase in the length or growth of bone. Undoubtedly, a good degree of venous stasis exists in a limb with an arteriovenous fistula; however, in analyzing our results we can say unequivocally only that we demonstrated increased filling of vessels, and an increased number of vessels filled by microangiographic techniques. These findings are associated with increase in length of bone and an increase in the recorded temperature of the femur.

SUMMARY AND CONCLUSIONS

The study was made on sixteen immature dogs in each of which an arteriovenous fistula was established in one hind limb. In these animals lengthening of the femur in the limb with the fistula was always associated with an increase in the surface temperature of the femur and with a consistent alteration in the blood supply of the femur.

Note: We acknowledge with gratitude the extensive contribution made by Dr. Julia Herrick, and her assistant, Glenn Christianson, to the work reported in this paper.

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Osseous Coccidioidal Granuloma

An Attempt to Measure the Prognosis *

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The diagnosis of coccidioidomycosis is established by culture of the organism *Coccidioides immitis*. If material for culture is not obtainable, then the diagnosis can be made by the combination of skin tests and complement-fixation tests. Localization of the focus of infection can be determined by the physical examination, roentgenograms, and recovery of the organism from the sputum, aspirate, or draining sinuses. The protracted course of the disease allows for important re-evaluations of significant clinical and laboratory findings.

The purpose of this paper is to present certain observations noted when the patients with osseous coccidioidomycosis were reviewed. Similarities, both clinical and laboratory, were noted in many of the cases. This afforded the authors an opportunity, without difficulty, to group various patients together in categories. These groups or categories are presented in an attempt to better understand the disease and its various phases. A prognosis also is considered; this is made possible by an appreciation of repeated clinical examinations and serial complement-fixation tests.

HISTORY

More than twenty years ago Carter, in a classic monograph, described the roentgenographic findings of osseous coccidioidal granuloma in seventy different cases. This report represented all of the cases seen up to that time at the Los Angeles County Hospital. Carter was first to emphasize the predilection for bone prominences and reported cases which involved all bones except the facial bones and all joints except the shoulder joint proper and the temporomandibular joint. He also compared the roentgenographic findings of coccidioidal infection of bones and joints with tuberculosis and infectious arthritis.

In 1939 McMaster and Gilfillan reviewed the clinical and pathological aspects of osseous involvement.

The outstanding contributions of Smith [7] and others to the interpretation of skin-test sensitivity and the complement-fixation tests have aided greatly in the diagnosis and prognosis of coccidioidomycosis.

In 1946, Forbus and Bestebreurtje reported a study of ninety-five cases seen in the Armed Forces over a five-year period. They believed that the disease occurs in two clinicopathological forms: (1) the primary pulmonary and (2) the secondary disseminated form. Of the ten most frequent sites of dissemination, bone ranked seventh.

Mazet was unable to observe any definite clinical pattern in the disseminated disease. He detailed examples of rapid dissemination and cases in which the clinical course was more leisurely and benign. Mazet observed that invasion of a single bone was unusual.

Recently, Birsner and Smart reported eighteen selected cases of chronic osseous coccidioidomycosis from Kern County, California. A high percentage of the

* Read, in part, at the Annual Meeting of the Western Orthopedic Association, Santa Barbara, California, October 20, 1957.

patients were well and employed when interviewed. The authors note a more favorable prognosis is afforded when only one or two bones are involved. In fifteen of their cases, there was a maximum of two lesions.

STUDY

This study was undertaken in an attempt to integrate the clinical, roentgenographic, and histopathological findings with the serial complement-fixation tests in order to determine the patient's prognosis at the time of initial evaluation.

In the ten-year period, 1946 to 1956, 331 cases of coccidioidomycosis were seen at the Los Angeles County Hospital. The charts of these patients were reviewed and those with osseous lesions set aside for careful study. Twenty-nine patients with coccidioidal granulomata of bone were encountered. Two new patients with osseous lesions were initially seen in the year of study (1956) and followed for a period of two years. A total of thirty-one patients was carefully evaluated. During the period of study, three of the initial twenty-nine patients came in for a long-term follow-up examination; a fourth experienced a recurrence of the disease and returned for treatment. One of the authors (M.B.) saw all of the thirty-one patients at one time or another during the course of their disease.

The diagnosis was established by isolation of the organism in culture in thirty of the thirty-one patients. In twenty-eight of the thirty, material for culture was obtained directly from the bone lesion or its draining sinus. In the remaining two sputum was cultured; these two had active pulmonary disease and later demonstrated pre-terminal osseous lesions. Diagnostic complement-fixation tests were positive to varying degrees in all of the thirty-one patients. In twelve of the thirty-one patients a biopsy specimen was obtained. During the study, these were reviewed with Dr. Edward Butt, Chief Pathologist of the Los Angeles County Hospital.

Careful analysis of the well documented cases revealed a number of significant factors important in evaluating the particular patient with coccidioidal granuloma. These factors are listed in order of considered importance:

1. The general condition of the patient, that is, the nutritional state, hemogram, and serum proteins;
2. Serial complement-fixation tests;
3. The duration of the disease, including, when possible, both the primary pulmonary and the disseminated phases;
4. The status of the primary pulmonary lesion determined by physical and roentgenographic findings;
5. The number of foci of dissemination recognized clinically and by roentgenographic skeletal surveys;
6. The histopathology, when available, from study of diagnostic biopsy material or material obtained from surgical intervention.

With respect to these factors, the original twenty-nine patients (and later the two additional cases) appeared to group themselves into three categories. Each group or category had consistencies of its own, that is, little variation noted in the six factors. The importance of time is emphasized. Time affords an opportunity to evaluate a patient periodically, both clinically and by laboratory measures, principally the complement-fixation test. However, in reviewing the records a definite impression as to the clinical course was obtained at the time of the initial examination. Because of this, patients could be grouped; and in two of the three categories a prognosis was possible. After presenting some general observations, these groups will be considered in detail.

Laboratory Procedures

The important role of the complement-fixation test in determining the prognosis in coccidioidomycosis necessitates some basic understanding of the test. It is performed in the same manner as the quantitative Wassermann or Kolmer test.

The antigen is made from a culture filtrate of the fungus *Coccidioides immitis* in Long's synthetic medium. The complement-fixation test consists of two antigen-antibody systems. The antibody consists of two systems, one the test and the other the indicator system. When the test is bound or fixed, no hemolysis occurs. This is recorded as a positive test. Conversely, when no antibody is present in the patient's serum the complement is not bound and lysis of the indicators system results.

The preparation of the antigen from a culture of the fungus in Long's synthetic medium is unpredictable. Several cultures set up simultaneously under identical conditions may yield only one antigen satisfactory for the complement-fixation test. Despite biochemical testing of numerous antigens no differences can consistently be observed to indicate the acceptability of antigens as distinct from one inadequate for complement-fixation tests.

The complement-fixation test has proved to be a valuable diagnostic and prognostic aid [8,9]. The 6,500 tests performed and recorded at the Los Angeles County Hospital confirm Smith's impression that the complement-fixation titer is parallel with the activity of the disease and not the degree of immunity. It has been observed that the test usually becomes positive by the fourth or fifth week after the onset of the disease and may, depending upon the amount of activity, remain positive for months or years. Every three to four months is an appropriate interval for checking the complement-fixation titer in chronic dissemination; whereas, monthly check-ups should be done in the primary pulmonary disease.

The serum of the patients is serially diluted in eight tubes, from 1/2 to 1/256. A titer positive in 1/2 or through four tubes (1/16) usually indicates primary pulmonary infection. Positive titers in the fifth or sixth tube (1/32 to 1/64) may represent dissemination beyond the confines of the lung. Further increases in the titer, with positive reading in the seventh or eighth tubes (1/128 to 1/256), are found with frank dissemination. Therefore, in disseminated disease with an active pulmonary lesion and multiple osseous foci the titers rise to the level of seven or eight tubes and remain high until death. However, when dissemination to a single site occurs the complement-fixation titer may be in the range of the primary pulmonary infection, or, at most, the fifth or sixth tube. With clinical improvement the titer may gradually fall over a period of months or years, but never precipitously.

The coccidioidin skin test is significant in the diagnosis of primary pulmonary disease. In the disseminated disease, that is, osseous coccidioidal granuloma, the skin test most frequently is negative. The complement-fixation test is by far the most valuable and specific for the disease.

General Information

The average age of the patients when first seen was twenty-nine years, the range being nine through seventy-three years. Racial predisposition was apparent. Twenty-seven of the patients were colored: twenty-two Negroes and five Indians. Women were in the minority: only four were seen in this series, three Negroes and one Indian.

A frank history of valley fever was obtained in nine; an additional ten patients resided in the San Joaquin Valley prior to, or at the time of, the onset of the disease. The remaining third of the patients came from widely scattered areas throughout California. Cotton-picking in Kern County was the most commonly stated occupation, although six patients denied handling soil or soil products. In patients with a history of valley fever evidence of osseous dissemination occurred within ten months, averaging three and a half months.

In nineteen of thirty-one patients, the initial complaints and physical findings were referable to the musculoskeletal system. These included pain, tumor, and soft-tissue draining sinuses of an extremity. Nine patients had complaints that were predominantly medical: chest pain, cough, weight loss, and skin lesions. Three patients had both medical and orthopaedic complaints.

An active primary pulmonary lesion was noted in twelve patients.

TABLE I

	General Condition	Complement Fixation	Duration of Disease	Pulmonary Lesion	Number of Foci	Histopathology
Group I	Very poor	High	Short	Active	Multiple	Suppurative
Group II	Fair	High	Short to long	Usually inactive	Usually single focus	Suppurative or proliferative
Group III	Good	Low	Short to long	Inactive	Single	Proliferative

Three-fourths of the patients reviewed had single osseous lesions. The incidence of a single focus increased with chronicity.

Two patients had tuberculosis as well. In one, the onset of pulmonary tuberculosis and the disseminated coccidioidomycosis appeared to be simultaneous. In the other, the patient acquired pulmonary tuberculosis after seven years of sustained coccidioidal infection of bone.

In forty-six skeletal lesions definite osseous predilections of this fungus were recorded. The spinal column (ten lesions) and the lower extremities distal to the knee (fourteen lesions) were favored sites of dissemination.

GROUPS IN DETAIL

The thirty-one patients with coccidioidomycosis of the bone were divided into three groups (Table I):

Group I, in which the patients were undergoing rapid fatal dissemination;

Group II, in which the patients appeared to be early in the course of the disseminated disease;

Group III, in which the patients demonstrated a single osseous focus of dissemination.

Group I: Rapid Fatal Dissemination (Eleven Patients)

1. *General condition:* The patients were severely debilitated by the disease with laboratory evidence of anemia, increased sedimentation rate, and a reversal of the albumin-globulin ratio.

2. *Serial complement-fixation titers:* All were positive through eight tubes (1/256). Nine of the eleven patients had high (1/256) titers when first seen. The other two rose to eight tubes with clinical dissemination (within three months).

3. *Duration of the disease:* Short duration. Dissemination occurred early, averaging two months. All but one patient died within thirteen months from the onset of the disease.

4. *Status of primary pulmonary disease:* The primary pulmonary lesion was active in eight of the eleven patients.

5. *Number of foci:* Approximately half of the patients had multiple osseous lesions, sometimes numbering five or six, while the remaining had only a single osseous focus. However, in the patients with a single osseous lesion, soft-tissue abscesses or pulmonary lesions, or both, were noted.

6. *Histopathology:* The histopathological study demonstrated a suppurative process with numerous spherules and endospores (Fig. 1).

O. S. (Case 16). A Negro man, forty-nine years old, was apparently well until December 1949, when fever, cough, and malaise associated with considerable weight loss developed. In January 1950, he was admitted to the Los Angeles County Hospital with a presumptive diagnosis of pulmonary tuberculosis. Chest roentgenograms revealed a large apical cavity. Sputum studies were positive for *Coccidioides immitis* and negative for acid-fast bacilli. The coccidioidin skin test was positive. On January 8, 1950, the complement-fixation titer was equivocal. On January 26, 1950, is was positive in two tubes (1/4).

He denied any complaints referable to the musculoskeletal system and no roentgeno-

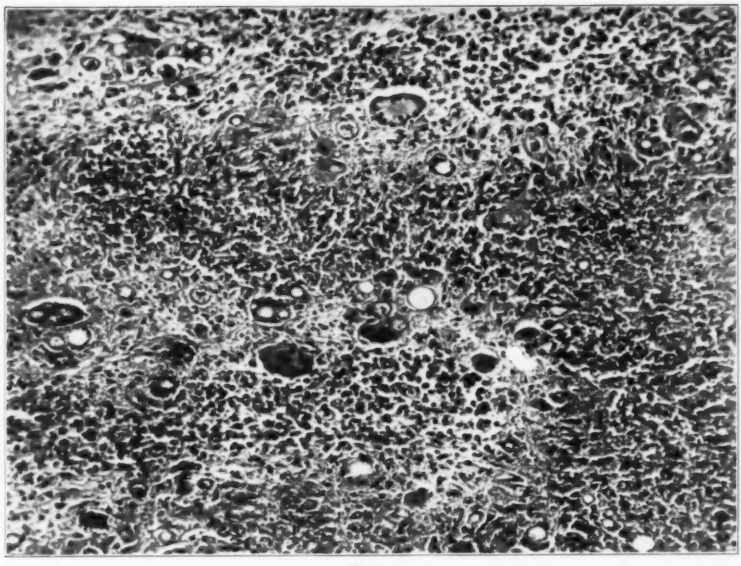

FIG. 1

An active suppurative lesion with numerous spherules, endospores, giant cells, and early proliferation.

grams other than those of the chest were ordered. He deteriorated rapidly. In February multiple cutaneous nodules developed, and on March 2, 1950, his complement-fixation titer was positive through eight tubes (1/256). He died the following week. At autopsy, osseous lesions were demonstrated in the right sternoclavicular joint and within the vertebral bodies of the first lumbar through the fourth lumbar vertebrae.

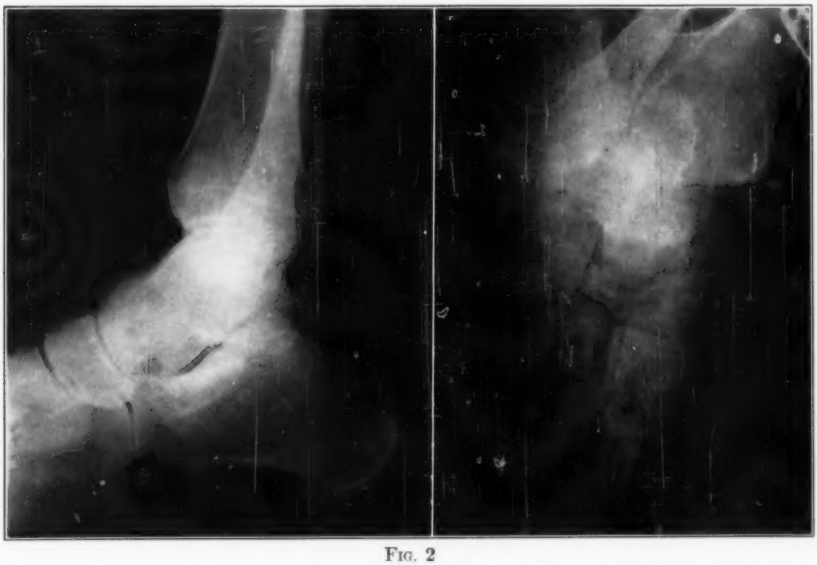

FIG. 2

J. J. Case 13. There was increased localized destruction, despite a drop in the complement-fixation titer. Three years separate the two roentgenograms (1953 and 1956). Originally the patient was classified in Group II. Later he was reclassified in Group III, and, because of persistent drainage, the leg was amputated below the knee.

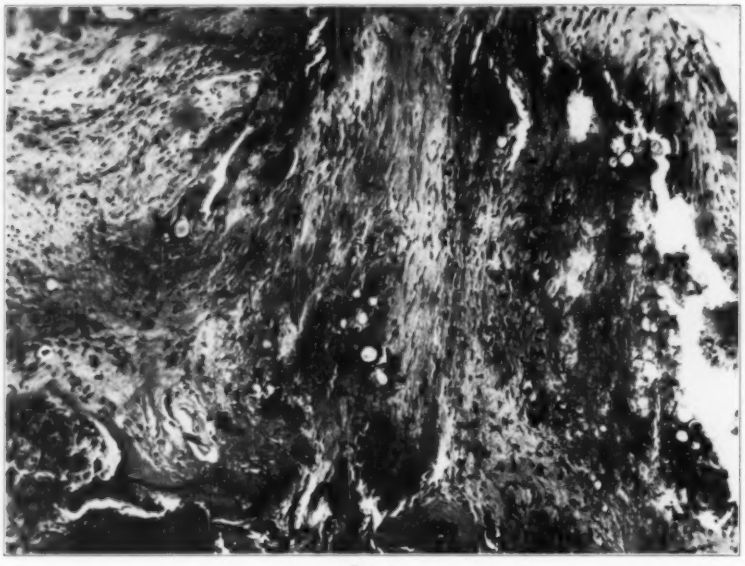

FIG. 3

Healing lesion with fibrosis and relatively few organisms. Evidence of old necrosis is to be seen at the far right.

Group II: Early in the Natural Course of the Disseminated Disease (Fifteen Patients)

1. *General condition:* The patients did not appear seriously ill and were not debilitated by the disease.

2. *Serial complement-fixation titers:* The complement-fixation titers, without exception, were positive in the disseminated range—six tubes (1/32) or higher.

3. *Duration of the disease:* These patients were first seen from one to twenty-eight months after the onset of the disease. They were relatively early in the course of disseminated disease, varying from one to eighteen months.

4. *Status of the primary pulmonary disease:* The primary pulmonary infection was inactive in eleven of the fifteen patients.

5. *Number of foci:* In eleven of the patients there was a single focus of dissemination (Fig. 2).

6. *Histopathology*: Considerable variation was noted, depending on the section obtained. The specimens studied varied from the suppurative to the proliferative fibrous-tissue phase (Fig. 3).

R. D. (Case 15), a thirty-year-old Negro woman was admitted to the hospital in January 1953 with swelling over the anterior aspect of the upper portion of the tibia. Three months prior to admission, a mass over the inferior aspect of the right breast had developed. This drained pus and slowly healed. A twenty-pound weight loss and a nonproductive cough were noted. Physical findings were limited to the left knee where a five-centimeter cystic mass was palpable. Roentgenograms revealed a lytic lesion of the tibial tubercle (Fig. 4). In February 1953, a block excision of the tibial lesion was done. Three hundred cubic centimeters of bloody purulent material was evacuated. On culture the organism was identified as *Coccidioides immitis*. The complement-fixation titer was positive through eight tubes (1/256). She was discharged three weeks postoperatively. The wound healed by secondary intention in six weeks. In June, her complement-fixation titer remained positive through eight tubes. In August, considered healed, she was discharged from care without the benefit of skeletal roentgenographic surveys and further complement-fixation tests. Attempts to locate this patient have not been successful.

Group III: Single Osseous Focus of Dissemination (Four Patients)

1. *General condition:* Clinically these patients did not appear ill and had no evidence of systemic disease.

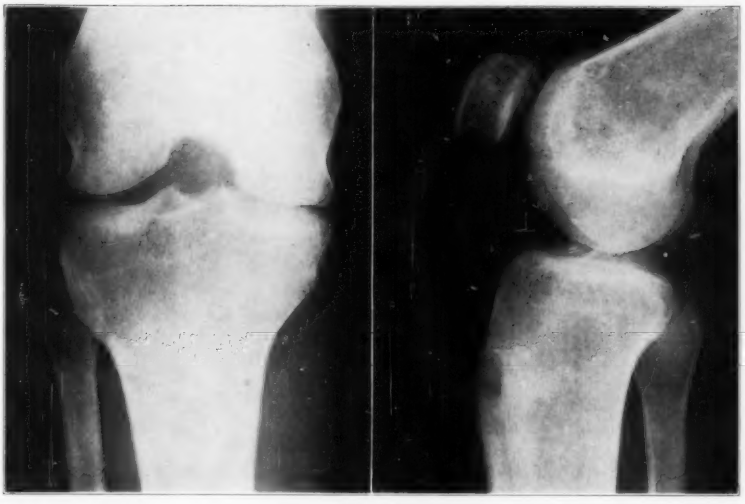

FIG. 4-A

FIG. 4-B

R. D. Case 15. Lytic lesion of the tibial tubercle. Fig. 4-A shows the preoperative condition and Fig. 4-B the result after surgical block excision.

2. *Serial complement-fixation titers:* In all four cases the complement-fixation tests failed to exceed titers beyond five tubes (1/32).

3. *Duration of the disease:* Variable. The history of the disseminated disease ranged from three to forty-eight months. In the patient with complaints of three months' duration, no history of a pulmonary process was obtained.

4. *Status of the primary pulmonary disease:* The primary pulmonary lesion was inactive in all four patients.

5. *Number of foci:* Single osseous focus of dissemination (Figs. 5 and 6).

6. *Histopathology:* The specimens demonstrated a proliferative phase with fibrous reaction and rare organisms (Fig. 7).

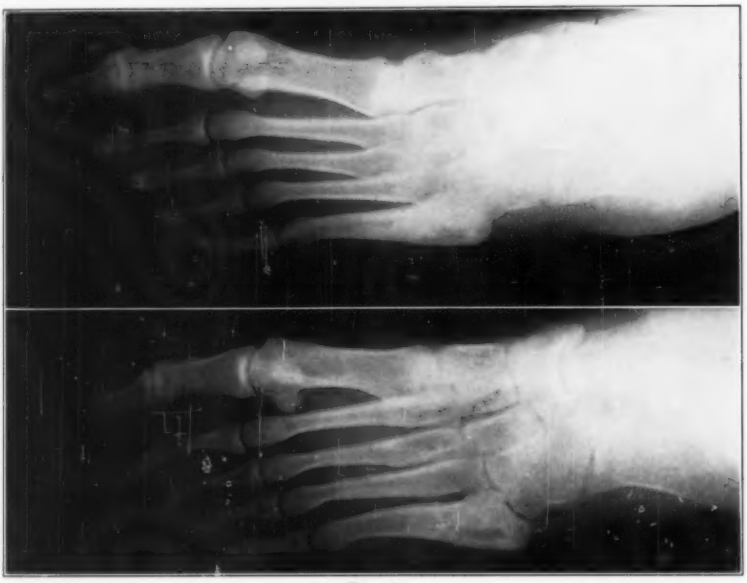

FIG. 5

D. H. Case 3. Single osseous lesion at the base of the fifth metatarsal responded well to treatment.

M. H. (Case 6), a Caucasian man, seventy-two years old, first noticed a mass in the distal third of the right forearm in September 1948. Roentgenograms made in November revealed an area of osteolytic destruction in the distal third of the ulna (Fig. 8). A biopsy specimen demonstrated coccidioidal granuloma. The complement-fixation titer was positive through five tubes (1/32). The lesion continued to drain. Because of excellent general health and the absence of a focus elsewhere, he underwent surgical resection of the distal half of the ulna. This healed by secondary intention in two months. He was last seen two and one-half years after the onset of the solitary focus of dissemination and was free of all drainage. His complement-fixation titer had decreased.

One patient was encountered who did not appropriately fit into any category. The patient had lived with his disease for ten years and demonstrated smoldering

FIG. 6

J. C. Case 27. A large destructive lesion of the fourth and fifth lumbar bodies. The histopathological picture was that of an active process. A spine fusion was done but the patient died within three months.

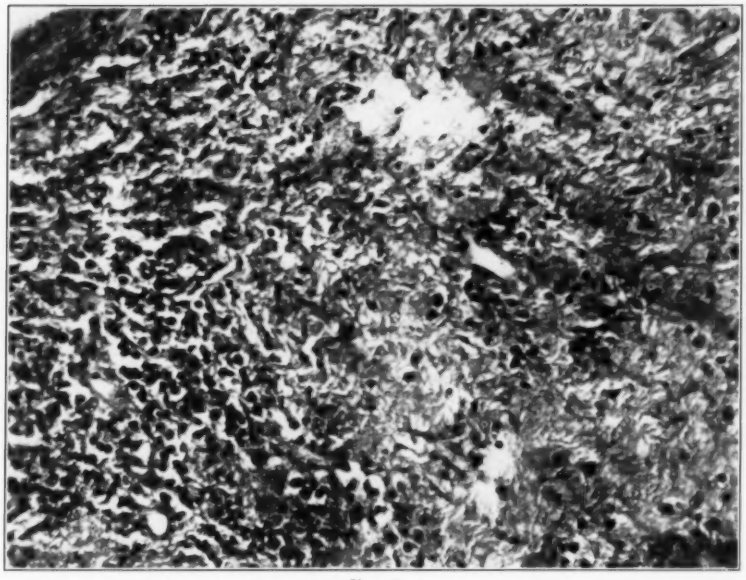

FIG. 7

This section reveals leukocytic infiltration with fibrous proliferation. However, organisms are rare; only one is seen in the lower left-hand corner.

activity with multiple lesions. The complement-fixation titers were positive through eight tubes (1/256).

W. M. (Case 26), a male Negro garage worker, forty-years old, had a diagnosis of coccidioidomycosis made in 1946. The right knee and left elbow were involved with multiple skin lesions. Numerous draining sinuses resulted. These eventually healed. In 1947 he began to experience episodes of back pain. His knee and elbow occasionally were painful but to no great degree. In 1956, he was admitted to the Los Angeles County Hospital with acute low-back pain. On admission, the patient appeared in good health with findings limited to the back, right knee, and left elbow. The physical findings of the back were consistent with an acute lumbosacral strain. Both the knee and elbow had mild effusions with associated thickening of the synovial membrane. The complement-fixation titer was positive (1/256). Roentgenograms revealed a lytic lesion in the proximal metaphysis of the tibia with no joint involvement. The elbow showed evidence of old healed osteomyelitis of the medial epicondyle of the humerus. Roentgenograms of the lumbar spine demonstrated rather severe degenerative arthritis rather well localized to the fifth lumbar and first sacral vertebrae (Fig. 9). The remainder of the skeletal survey was normal. The diagnosis of degenerative-disc disease was made. He responded promptly with conservative management and was free from recurrence on follow-up visits. The findings in his knee and elbow remained unchanged.

DISCUSSION

This study was undertaken in an attempt to determine the prognosis of any patient with osseous coccidioidal granuloma. A prognosis is necessary before any consideration of treatment. The desire to establish a prognosis, and thus to treat accordingly, motivated the authors to review the cases at the Los Angeles County Hospital.

The high incidence of osseous lesions, nearly 10 per cent of the total cases of coccidioidomycosis, proved surprising. Perhaps this represents a sampling variation; relatively more patients with disseminated disease were seen.

It became apparent in the study that the patients with clear-cut clinical findings, permitting a prognosis when first seen, rarely demonstrated subsequent need for revision of the prognosis (Table II). This was characteristic of Groups

TABLE II

Original Group Classification	No. of Cases	Observations during Course of Disease	Final Group Classification	No. of Cases
Group I	11	10 men: death 1 woman: improved; reclassified Group II	Group I	13
Group II	15	3 deaths: Group I 4 improved: Group III 9 unchanged (inadequate follow-up)	Group II	10
Group III	4	1 death: Group I 3 well after surgery	Group III	7
Total	30*		Total	30*

*One patient did not appropriately fit into any category.

I and III in which the prognosis in only two patients was altered. The predictable fatalities in Group I were Negro men, all dying within thirteen months. Patients in Group III also demonstrated a predictable outcome, but, in sharp contrast, the prognosis was good. In Group II, a temporary category, the prognosis in six of the original fifteen patients was altered. These six patients were observed for an average of four years while the remaining nine patients were followed for an average of eleven months. If the period of observation in these latter patients had been adequate, then there would have been revision of the prognosis in these.

The sole exception to the fatal outcome of Group I was a Negro female who was alive six years after onset. It is most interesting to conjecture about sexual differences in the outcome of particular patients within a certain group. In general women show less susceptibility (only four of the thirty-one patients were women) and greater resistance or tolerance in handling the infection (no deaths). Therefore, it would appear from this study that the ominous prognosis is in an acutely

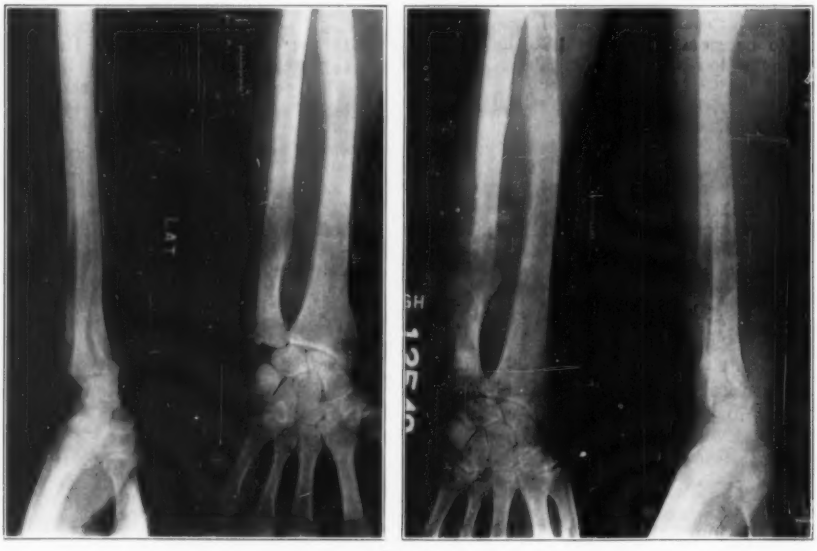

FIG. 8

M. H. Case 6. Roentgenograms show a lytic lesion of the distal portion of the ulna. The roentgenogram on the left was made November 1948, that on the right two months later. The destruction increased without apparent rise in the complement-fixation titer.

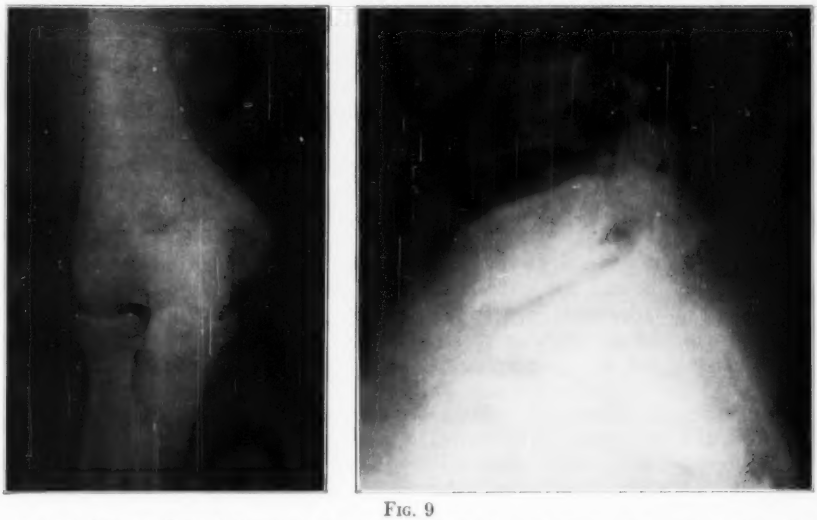

FIG. 9

W. M. Case 26. Healed disease of the distal portion of the humerus and marked narrowing of the lumbosacral joint. The latter lesion may represent traumatic degenerative disc disease and not destruction secondary to disseminated coccidioidomycosis.

ill Negro man who is seen with primary pulmonary disease which, within one to three months, goes on to widespread dissemination. In our final analysis (Table II) thirteen of thirty-one patients (45 per cent) died as a result of disseminated disease. Thus, contrary to previous concepts, disseminated coccidioidomycosis is not universally fatal.

In Group I, eight of the eleven patients had persistent pulmonary lesions as compared with a total absence of pulmonary activity in patients in Group III. Those patients with a single chronic osseous lesion (Groups II and III) frequently were unable to recall symptoms of the primary pulmonary disease and the absence of this clear-cut history may in itself be a good prognostic sign.

The value of the complement-fixation test for the diagnosis arises from certain characteristics of the disease, that is, little available sputum and difficulty in culturing the organism, and the close resemblance of the disease to tuberculosis. During the primary pulmonary disease the proof of the diagnosis lies in the skin test and the complement-fixation test, by conversion (negative to positive) of the skin test, and the range of the complement-fixation titer (1/16). In the disseminated disease material for culture of the organism is usually available. The titer in patients with severe or widespread disease is 1/128 or 1/256. The advantage of serial complement-fixation tests is that a drop in the titer accompanies the focalization of the disease even though the activity is still vouched for. An increase in the severity of the disease, or an increase in the local destruction, may be indicated by a rising titer. Even though an initial titer was high, indicating dissemination, a persistent low titer will gradually develop in a patient with a single extrapulmonary focus. Fear of spread elsewhere from the single focus is limited, but continued activity (indicated by the complement-fixation titer) even in a solitary site may extend over a period of years. This smoldering infection may, and fortunately often does, burn itself out. The cessation of drainage of the sinus may be encouraging, but the true indication of activity is the continued low titer of the complement-fixation test. The titer parallels the protracted periods of months or years and the variable remissions and exacerbations. The usefulness of the complement-fixation test extends beyond osseous lesions and may well indicate dissemination to areas not visualized on roentgenograms, that is, liver and spleen. In essence, the examin-

ing physician should take a long careful clinical look at the patient and then verify his impression with the specific complement-fixation test.

The skin test in disseminated disease is of limited value. By the time dissemination occurs the skin test is frequently negative in the dilutions used. Although a 1/10 dilution of coccidioidin is sometimes helpful in achieving a positive reaction, cross reaction with other fungus antigens may occur at this concentration. The intrinsic wide variations within the techniques of administering and reading the test make its value more confined than the complement fixation test. A negative skin test can lead to disregarding the possibility of disseminated coccidioidomycosis.

We feel that skeletal surveys are more important than study of available biopsy material in evaluating the patient and ascertaining a prognosis. The two patients in this series with apparent single osseous lesions, but high complement-fixation titers, did demonstrate foci elsewhere when surveyed roentgenographically. Skeletal surveys are prerequisite in considering the ablation of an area thought to represent a single focus. This study verifies the conclusion of Birsner and Smart that the prognosis proved more favorable in patients with a single osseous lesion. However, one must substantiate the fact that the focus is singular.

Histopathological study when specimens are available from biopsy, although it may be diagnostic, has limited value in determining prognosis. Of the factors considered, study of a biopsy specimen was the most variable because it depended on the area studied. A certain degree both of suppuration and proliferative fibrous reaction was noted in all the osseous lesions. Generally, endosporulation is noted with suppuration, but not infrequently considerable marginal fibrous proliferation is also observed. Care must be taken to study more than one section for the most prominent histopathological picture is the prime consideration in coccidioidomycosis.

Of the cases documented three were classic of the respective groups.

O. S., Group I, demonstrated five unequivocal factors: (1) a sick patients, (2) a complement-fixation titer which rose from equivocal to positive through eight tubes (1/256) in eight weeks, (3) a disease of short duration—sick one month and dead within three months, (4) an active pulmonary lesion, and (5) multiple disseminated lesions. The prognosis in this case was poor.

R. D., Group II, was observed for a short time; the prognosis was undetermined. The patient did not appear to be particularly ill despite weight loss and two lesions—one osseous and the other soft tissue (breast). Surgery was done despite a complement-fixation test with a titer positive through eight tubes (1/256). Fortunately, the procedure resulted in local healing, but there was no satisfactory follow-up. Skeletal surveys were clearly indicated in this patient because she may have had other osseous lesions. Surgery should have been postponed until her complement-fixation titer dropped. She must remain in Group II (undetermined prognosis).

M. H. was certainly characteristic of Group III. This patient, although elderly and with considerable local destruction, was in excellent health. The complement-fixation titer was low (1/32) and the focus was clinically determined to be single. The prognosis was good; surgery resulted in healing. The patient was followed for two years.

Surgery is frequently contemplated in cases of osseous coccidioidal granuloma in an attempt to provide complete healing. The surgeon must remember that plaster immobilization has been effective and should be considered. The various surgical procedures might consist of amputation, exenteration of the focus, or arthrodesis of one or more joints. Contemplation of surgery necessitates complete evaluation of the patient and the determination of the prognosis. From the observations in this study surgical measures do not seem warranted in those patients in poor health with high or rising complement-fixation titers and multiple lesions, with or without pulmonary activity. This eliminates consideration

of surgery for patients in Groups I and II; only symptomatic and supportive treatment seems indicated.

Surgery was eventually done in ten patients; it was successful in eight and failed in two. Seven of the eight patients in whom surgery was successful were in Group III at the time of the surgery. The eighth patient was in Group II (R. D., Case 15). The failures are understandable; one patient (Group III) died of dissemination following surgical complications, the other patient (Group II) was subjected to guillotine amputation because of a large extremity lesion, secondarily infected. However, surgery done when indicated and compared with drug therapy is, to date, the most successful definitive form of treatment.

One of us (M. B.) has watched with interest the various forms of medical treatment in the last twelve years. The disease is so unpredictable that a five-year period of time is required to evaluate any drug. Fiese, in an excellent monograph, summarized sixty years of various forms of treatment as follows:

"1. The course of acute fulminating disseminated coccidioidomycosis has never been altered by any form of therapy, past or present.

"2. Meningitis has also probably not been affected by treatment, at least not often enough to draw any conclusions.

"3. Dramatic improvement of chronic disseminated lesions has co-incided with treatment by several methods and with many drugs.

"4. Drugs which have apparently been curative in some cases have failed in other similar cases.

"5. Apparent eradication of disseminated lesions may be followed ten or fifteen years later by severe or fatal recurrence."

Our observations concur with those of Fiese. Recently amphotericin B has proved popular in the treatment of the disseminated disease. We are without experience with amphotericin B.

CONCLUSIONS

1. Thirty-one patients with osseous coccidioidal granuloma were reviewed. The six factors considered important in evaluating the particular patient with coccidioidomycosis of the bone were:

a. The general condition of the patient, that is, the nutritional state, hemogram, and serum proteins.

b. Serial complement-fixation tests.

c. The duration of the disease, including, when possible, both the primary pulmonary and the disseminated phases.

d. The status of the primary pulmonary lesion determined by physical and roentgenographic findings.

e. The number of foci of dissemination recognized clinically and by roentgenographic skeletal surveys.

f. The histopathology, when available, from diagnostic biopsy material or material obtained at surgical intervention.

Three groups of patients were established, each with an apparent prognosis, one grave (Group I), one good (Group III), and one requiring time for any such determination (Group II).

2. No form of therapy, medical or surgical, alters the course of acute fulminating disseminated coccidioidomycosis (Group I).

3. Surgical measures may be indicated in Group III, but not in Groups I or II. However, when surgery seems warranted there is no need for hurried accomplishment.

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Parosteal Lipoma

A Report of Two Cases with Associated Bone Changes

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It is the purpose of this paper to discuss bone changes associated with benign, deeply seated, parosteal lipomata of the extremity. To our knowledge,

Fig. 1-A

Anteroposterior and lateral roentgenograms of the left leg revealed the radiolucent, circumscribed mass in the left calf and widening of the interosseous space. Note the sclerosis in the tibia and fibula adjacent to the soft-tissue tumor.

these bone changes have been reported but once in the literature in the last three decades[1]. The term *parosteal lipoma* was originally used by D'Arcy Power, in 1888, because the tumor was located next to the bone. The term *periosteal lipoma*, subsequently employed by Bartlett would seem to imply that the lesion arose from periosteum although fat cells are not present in the cambium layer of the

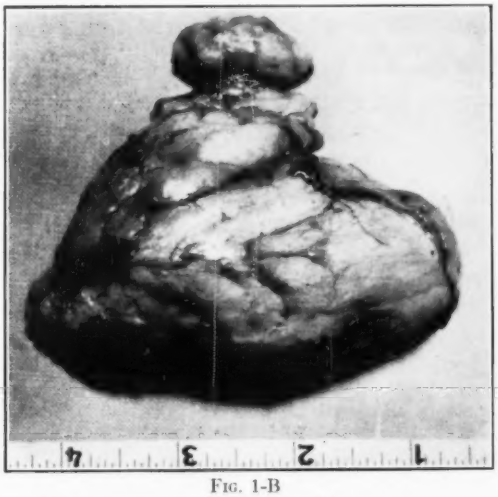

FIG. 1-B

The lipomatous mass removed *in toto*.

FIG. 1-C

FIG. 2-A

Fig. 1-C: Lateral roentgenograms, made two years postoperatively, revealed normal soft-tissue density, mild residual bowing of the tibia and fibula, and absence of sclerosis of these bones.

Fig. 2-A: Lateral roentgenogram of the left femur revealing the radiolucent, circumscribed mass in the mid-third of the left femur, with bone excrescences and sclerosis of the base.

periosteum. Fairbank and Richmond used the term *parosteal lipoma* in 1953. Therefore, we have continued the use of the term *parosteal lipoma* so as not to confuse the terminology.

Case 1. G.E., a four-year-old white girl, was brought to us on March 21, 1957, because of swelling of the left calf, which had been noted by a neighbor who called it to the attention of the parents. There were no symptoms. The past history was non-contributory.

The child was well developed and walked with a normal gait. There were no abnormalities of the upper extremities, trunk, or spine. The left calf showed diffuse enlargement but no distinct palpable mass; it was three-quarters of an inch larger than the right in circumference. There were no areas of tenderness. Elevation of the extremities did not cause any change in

Fig. 2-B

The lipomatous tumor removed. The arrows point to the bone base.

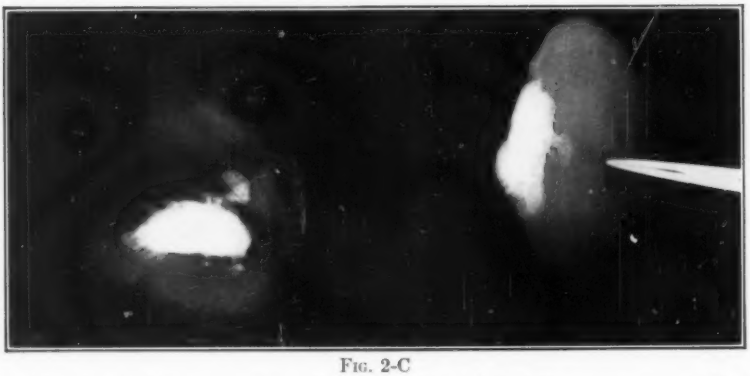

Fig. 2-C

Roentgenograms of the removed specimen reveal the detail of the excrescences at the base of the lipomatous mass.

the size of the calf. Muscle function and sensation were within normal limits. Roentgenograms of the left leg (Fig. 1-A) revealed an area of decreased density about four and a half inches long and two inches wide in the posterior compartment of the leg. There was anterior bowing of the tibia and irregularity of the mid-portion of the fibula. Moderate sclerosis of the cortices of the tibia and fibula in the middle third was present. Widening of the interosseous space was also noted. A preoperative diagnosis of parosteal lipoma was made.

On March 27, 1957, with the patient under general anesthesia and tourniquet control, the left calf was explored. An incision, the full length of the calf, was made just medial to the tibia.

The gastrocnemius and soleus muscles were retracted posteriorly; a yellowish tumor, filling the entire posterior compartment of the leg, was visualized. The tumor had penetrated the interosseous membrane, with tumor tissue presenting in the anterior compartment. The opening in the interosseous membrane was enlarged in order to remove the tumor *in toto* (Fig. 1-B). It was noted that the tibia was bowed and the interosseous space was widened. The gastrocnemius and soleus muscles were displaced posteriorly. The tibialis posterior muscle was displaced medially, whereas the flexor hallucis muscle was displaced laterally and inferiorly. Pathological examination revealed a lipoma with mature fat cells. At the last follow-up examination on March 20, 1959, the clinical findings were entirely normal. Lateral roentgenograms of the legs (Fig. 1-C) showed only slight residual bowing of the tibia. The sclerosis was completely gone.

Case 2: K.D., a white man, forty-nine years old, was seen on June 4, 1956, for a lumbosacral derangement without nerve-root irritation. Incidental to the examination, a large mass extending over the left thigh in its middle third was discovered. The mass, approximately four and a half inches in length, was firm and fixed to the underlying bone. A lateral roentgenogram (Fig. 2-A) of the involved area revealed a soft-tissue mass of extreme radiolucency over the anterior medial aspect of the middle one-third of the femur. The mass appeared to arise from bone. Several bone exerescences projected from the femur into the radiolucent area. At the base of these projections there was some sclerosis of the femoral shaft which was associated

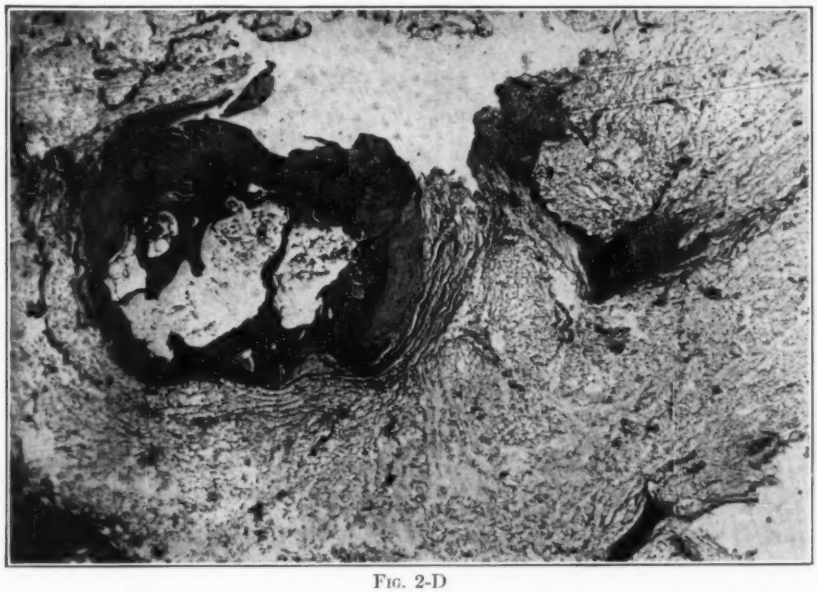

Fig. 2-D

Low-power microscopic section of the specimen, revealing the thickened periosteum, without inflammatory cell infiltration, and sclerotic bone at the base of the tumor with the surrounding lipoma (× 80).

with some irregularity of the surface of the bone cortex. A tumor, three inches in diameter, was removed on August 15, 1956, through a six-inch incision over the anterior medial aspect of the left thigh. The bone pedicle was removed with the mass (Figs. 2-B and 2-C). There was no evidence of inflammation of the bone or periosteum. The wound was closed in layers. Histological examination of the specimen revealed sclerotic bone trabeculae surrounded by adult fat cells with intercellular connective tissue. The periosteum was thickened but contained no inflammatory cells (Fig. 2-D). The postoperative course was uneventful. Clinical examination subsequently (May 15, 1957) has not revealed any recurrence of the lesion or any roentgenographic abnormalities.

DISCUSSION

As demonstrated in the first case reported here and in the similar case reported by Bartlett, a large lipomatous mass in close relationship to a growing

bone may cause deformity. In the second case presented, the bony exostosis which arose from the anterior aspect of the femur in its middle third was surrounded by the lipomatous tumor. We believe that these bone changes were related to the surrounding lipoma. The location of the bone excrescence in the center of the femur is not the usual site for an osteoma or an osteochondroma. Furthermore, there was no cartilaginous cap, but rather a lipomatous cap. For these reasons we believe that the bone spur formed as a result of stimulation of the periosteum by the overlying tumor.

Lipomata do not produce symptoms unless they are large enough to restrict joint motion or to compress a vital structure, such as an artery, nerve, or lymphatic vessel. The mere presence of a lipoma near a vital structure does not necessarily produce symptoms. In this connection, Fairbank reported a parosteal lipoma in the vicinity of the posterior interosseous nerve. There was a full range of motion of the elbow and no evidence of paralysis. Richmond, on the other hand, reported a case in almost the identical location, but in this instance there was pressure on the posterior interosseous nerve and partial paralysis of the extensors of the fingers and wrist. Although these tumors arose from periosteum, there were no bone changes seen on roentgenograms, but radiolucent masses were demonstrable in the soft tissues. Robson described one of the largest lipomata—a tumor weighing 5,200 grams—with extensive calcification in the thigh. The lesion produced irreparable obstruction of major vessels, and ablation was necessary.

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Biochemical Studies of Articular Cartilage

III. Values Following the Immobilization of an Extremity *

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As a continuation of our work on atrophy of articular cartilage a complementary study on simple disuse atrophy was logical. A better understanding was needed of the similarities as well as the differences in the metabolism of atrophic and normal cells. An investigation similar to the one previously done on denervation atrophy [1] therefore was performed, using experimental immobilization of an extremity to produce the disuse atrophy. The effect of immobilization atrophy on the metabolism of skeletal muscle has already been presented [7]. This study on articular cartilage was carried out simultaneously with the one on skeletal muscle.

This report gives data on two groups of puppies arranged according to age and length of time of immobilization: Group I: seventy-eight to ninety-eight days of age with twenty-eight to forty-one days of immobilization; Group II: ninety-nine to 124 days of age with thirty-five to fifty-eight days of immobilization.

PROCEDURES AND METHODS

The puppies were bred by us and at designated days of age were subjected to an internal immobilization of one hind leg as follows: With stainless-steel wire the proximal third of the femur was wired to the distal portion of the tibia and the distal third of the femur to the wing of the ilium. The wires were passed around the long bones and through a hole in the ilium. After acutely flexing the hip and knee, the wires were tightened, fixed, and buried. Skin closure alone was performed. The puppies had good locomotion on three legs.

The puppies were killed at different ages and at predetermined lengths of time of immobilization and the articular cartilage obtained from both the control and from the immobilized extremity. The tissues were taken from all puppies under nembutal anesthesia. Details of the techniques for the removal of serum and the cartilage and for the preparation of the tissues for chemical analyses as well as the chemical methods used have already been presented [4,6,8].

The determinations made on the serum were water, chloride, sodium, potassium, and total nitrogen while on the cartilage water, chloride, total nitrogen, collagen nitrogen, sulphate, sodium, and potassium were determined. Several times the sodium and potassium determinations on the experimental cartilage had to be omitted because the amount of cartilage obtainable from the immobilized limb was only about 75 per cent of that obtained from the weight-bearing limb.

Since this study parallels our work on denervation atrophy, the same symbols and methods of calculation adopted previously [8] are used. Parentheses ( ) represent milliequivalents or grams per kilogram of tissue; brackets [ ] represent milliequivalents or grams per kilogram of tissue water; and braces { } represent milliequivalents or grams per kilogram of phase or compartment.

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Briefly, cartilage is a specialized form of connective tissue composed of chondrocytes in an extracellular compartment. The chondrocytes consist of water and solids. The extracellular compartment has a fibrillar structure, the fibers being mainly of collagen. Between these fibers is a fluid like all other extracellular fluids in composition together with an amorphous ground substance characterized by a mucopolysaccharide, chondroitin sulphate. Thus the extracellular compartment of cartilage consists of three subphases: (1) the connective-tissue phase containing fiber solids and some extracellular fluid, (2) the chondroitin-sulphate phase which is in the spaces between the collagen fibers, and (3) the extracellular fluid which is an ultrafiltrate in the interstices. The tissue fluid and the ground substance are considered to be interconnected throughout.

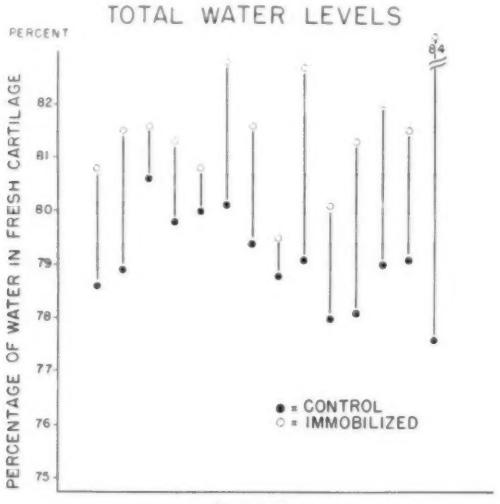

CHART I

The assumptions which are pertinent here and have been stated previously are:

1. The connective tissue of cartilage resembles all other connective tissues in water, nitrogen, and electrolyte concentrations. As a consequence, we have judged that the connective-tissue phase in cartilage resembles that of the dense connective tissue, tendon, and that the collagen nitrogen of cartilage exemplifies the total connective-tissue weight in cartilage. This concept was based on the work of Manery, Danielson, and Hastings.

2. The amounts of water, chloride, sodium, and potassium identified with the connective-tissue phase of cartilage correspond with those found in the dense connective tissue, tendon.

3. Under normal metabolic conditions the chondrocytes of cartilage contain no chloride. In 1956, Brower using a silver nitrate stain for chloride as described by Gersh showed by qualitative histological identification that the dense collection of silver oxide granules is in the extracellular matrix immediately adjacent to the chondrocytes. No granules were seen within the chondrocytes. Therefore, using these assumptions we have calculated the mass of each of the compartments in a kilogram of cartilage. The chloride values in the tissue indicate the volume of extracellular fluid, the collagen nitrogen values the weight of the connective-tissue solids (solids in the connective-tissue phase) and the sulphate values the weight of the chondroitin sulphate, leaving the remainder

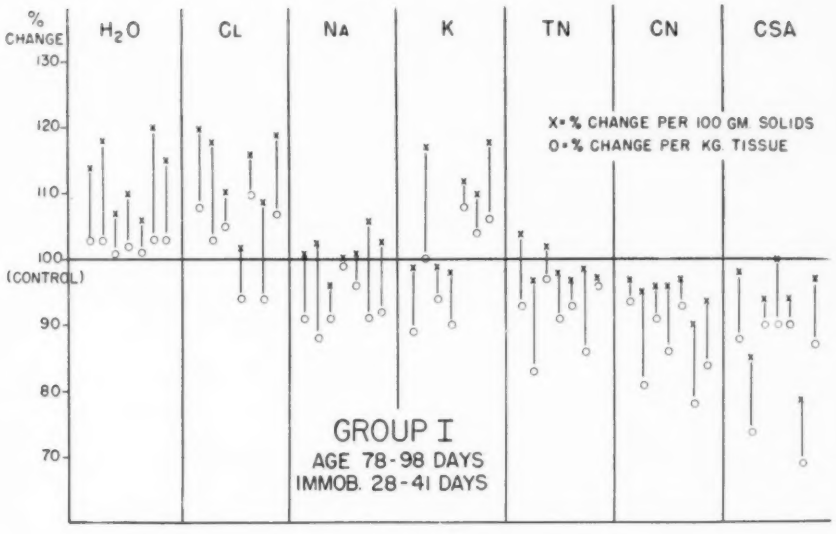

CHART II

of the kilogram of tissue to be allocated to the weight of the chondrocytes.

The simplest approach to obtain derived data from the original chemical analyses expressed in units per 100 grams of cartilage solids is to derive first the grams of connective-tissue solids or fiber solids in the 100 grams of cartilage solids. This is done by using the fact that 100 grams of tendon solids are associated with 15.8 grams of collagen nitrogen. Therefore, the amount of collagen nitrogen in cartilage represents a proportionate weight of fiber solids or connective-tissue solids.

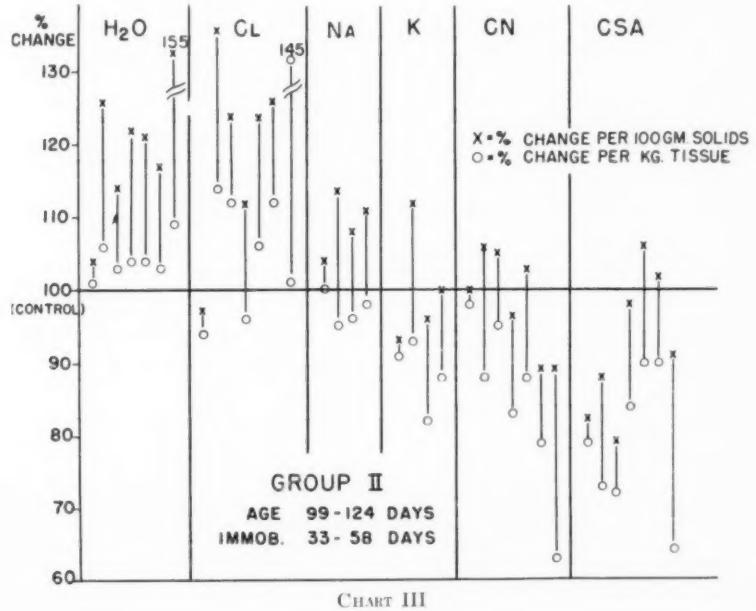

CHART III

TABLE I *†

VALUES PER 100 GRAMS OF ARTICULAR CARTILAGE FROM PUPPIES. ORIGINAL DATA

	Dog (*No.*)	Age (*Days*)	Immob. (*Days*)	H_2O (*gm.*)	Cl (*mEq.*)	Na (*mEq.*)	K (*mEq.*)	Total Collagen N (*gm.*)	Total Collagen N (*gm.*)	Total Collagen CSA (*gm.*)
Group I: 78–98 days of age. 28–41 days of immobilization										
Tissue: serum	22	95	28	932.1	109.5	147.5	5.39	7.00		
Cartilage										
Control hind limb				786.0	31.80	88.0	11.63	12.62	10.05	13.1
Immobilized hind limb				807.8	38.20	88.9	11.50	13.11	9.80	12.9
Tissue: serum	23	98	31	928.6	112.4	149.8	5.04	8.17		
Cartilage										
Control hind limb				788.7	34.15	94.3	11.09	13.83	9.84	11.5
Immobilized hind limb				815.0	40.30	97.2	12.98	13.50	9.32	9.8
Tissue: serum	28	78	30	930.8	111.6	146.2	4.95	8.00		
Cartilage										
Control hind limb				806.5	34.30	130.5	13.35	12.30	8.26	18.0
Immobilized hind limb				816.2	37.60	125.2	13.21	12.66	7.94	16.9
Tissue: serum	29	82	34	934.2	110.7	146.2	5.24	7.31		
Cartilage										
Control hind limb				798.0	34.45	119.8	11.97	12.88	8.73	16.7
Immobilized hind limb				813.0	35.10	120.3	11.72	12.65	8.42	17.1
Tissue: serum	30	85	37	930.6	112.0	141.6	5.02	8.00		
Cartilage										
Control hind limb				800.0	29.46	96.1	11.09	12.97	8.76	16.1
Immobilized hind limb				808.5	34.10	96.7	12.47	12.57	8.47	15.1
Tissue: serum	31	89	41	934.4	113.0	147.5	5.00	7.49		
Cartilage										
Control hind limb				801.0	36.44	107.3	9.92	12.60	8.24	15.2
Immobilized hind limb				827.8	39.76	113.4	11.95	12.54	7.42	12.1
Tissue: serum	11	87	28	925.6	109.6	144.2	4.82	8.65		
Cartilage										
Control hind limb				794.2	34.18	102.6	10.83	13.33	9.05	15.1
Immobilized hind limb				815.5	40.76	105.0	12.76	12.95	8.51	12.1
Group II: 99–124 days of age. 33–58 days of immobilization										
Tissue: serum	10	99	49	931.0	115.8	146.5	4.65	7.73		
Cartilage										
Control hind limb				788.5	32.50	90.9	12.36	12.98	8.66	15.0
Immobilized hind limb				794.8	31.57	94.2	11.56	13.03	8.70	12.3
Tissue: serum	24	102	33	930.0	112.2	144.0	5.00	8.31		
Cartilage										
Control hind limb				790.8	33.37	86.04	10.23	13.75	9.44	9.8
Immobilized hind limb				826.8	45.97	98.43	11.50		10.00	8.6
Tissue: serum	25	108	38	931.6	111.0	144.5	4.88	7.86		
Cartilage										
Control hind limb				780.0	30.90	78.6	10.22	13.42	9.90	12.9
Immobilized hind limb				801.5	38.30				10.40	9.
Tissue: serum	26	111	44	929.2	109.0	144.1	4.82	8.11		
Cartilage										
Control hind limb				781.0	30.95	78.54	10.58	13.64	10.42	11.3
Immobilized hind limb				813.0	34.70	85.2	10.16	13.32	10.10	11.1
Tissue: serum	20	117	47	933.9	117.5	149.0	4.62	8.12		
Cartilage										
Control hind limb				790.5	33.20	78.4	9.05	12.60	9.00	10.0
Immobilized hind limb				820.6	41.10				9.31	10.6
Tissue: serum	12	118	58	928.0	113.4	147.5	4.62	8.30		
Cartilage										
Control hind limb				791.2	34.54	91.9	11.05	13.30	9.67	10.4
Immobilized hind limb				815.5	43.61	102.0	11.00		8.64	10.6
Tissue: serum	21	124	47	927.1	109.6	146.0	4.83	8.75		
Cartilage										
Control hind limb				775.8	29.63	78.4	9.71	12.54	10.20	10.5
Immobilized hind limb				843.2	42.95				9.12	9.6

* Serum values are given in units per kilogram of serum.

† Cartilage values are given in units per 100 grams of cartilage solids except water values which are given in grams per kilogram of fresh cartilage.

The next step is to determine the weight of the chondroitin sulphate. The mass of chondroitin sulphate per 100 grams of cartilage solids is calculated from the millimol of sulphate obtained after the hydrolysis of the cartilage solids, using the molar weight of chondroitin sulphuric acid derived by Levene. Therefore, the sum of these first two steps will give the weight of the extracellular solids per 100 grams of cartilage solids, leaving the remainder of the 100 grams to be allocated to the chondrocyte solids.

Since the fibers of cartilage are similar in water, nitrogen, and electrolyte concentrations to other connective tissues (we have used tendon), estimations for the amounts of water, chloride, collagen nitrogen, sodium, and potassium associated with the weight of the fibers in the cartilage can be made. From previous studies, 100 grams of tendon solids are associated with 157 grams of water, 15.8 grams of collagen nitrogen, 20.65 milliequivalents of chloride, 20.2 milliequivalents of sodium [3], and 2.2 milliequivalents of potassium [15].

RESULTS

The original data obtained from the analyses of serum and articular cartilage are given in Table I. Since the puppies differed in age as well as in length of time of immobilization, the data for each puppy are given. The principal difficulty in making chemical analyses of articular cartilage is the small amount of tissue obtainable, which limits the extent of any study. Not only does the amount of cartilage normally decrease with the age of the animal, but this amount is further reduced in joints with experimental atrophy.

Although the original data have been given in units per 100 grams of cartilage solids with the exception of the total water, these values can be converted to units per kilogram of fresh cartilage simply by multiplying these values by the number of 100 grams of solids per kilogram of fresh cartilage. When one considers the original data, only general indications emerge and these can best be discerned in charts. In Chart I, the percentage of total water in the control fresh cartilage (black circles) and in the immobilized cartilage (clear circles) indicates that during atrophy there has been a distinct volume increase in the total water of the articular cartilage in both groups of animals. The percentage change in the other constituents in the two groups of animals is indicated in Charts II and III. The control cartilage value was regarded as the normal for each puppy, and this value has been considered 100 per cent. The immobilized values were therefore calculated as percentage of change from the control values. This change, using 100 grams of cartilage solids as the basis, is indicated by $\times$ on the chart. With the use of a kilogram of fresh cartilage as the basis, the percentage of change is indicated by a circle.

In both groups of animals, irrespective of whether the percentage of change was considered from the solid values or from the fresh cartilage values, the major changes were:

1. A percentage increase in the total water content with a larger percentage increase in the solids;

2. A percentage increase in chloride content with a larger percentage increase in the solids;

3. A percentage decrease in total nitrogen, collagen nitrogen, and chondroitin sulphate with a greater percentage decrease in the fresh cartilage.

Chemical analysis of a tissue does not portray its histological compartments (extracellular and intracellular). Rational treatment of the data must be carried out with calculations by deduction, a method which is generally known as the quantitative histochemical approach. Therefore, from our analytical data we have first calculated the mass of the extracellular and the mass of the intracellular solids in 100 grams of the total cartilage solids. These calculations are shown in Table II, considering the extracellular solids to be the sum of the connective-tissue solids and the chondroitin sulphate, and the intracellular solid

TABLE II *

HISTOCHEMICAL DATA FOR ARTICULAR CARTILAGE SOLIDS

Estimated Weight of Extracellular (E)$_s$ and Intracellular (C)$_s$ Solids in 100 Grams of Cartilage Solids

	Dog (*No.*)	Age (*Days*)	Immobilized (*Days*)	(F)$_s$ (*gm.*)	CSA$_s$ (*gm.*)	(E)$_s$ (*gm.*)	(C)$_s$ (*gm.*)
Group I: 78–98 days of age, 28–41 days of immobilization							
Cartilage	22	95	28				
Control hind limb				63	13	76	24
Immobilized hind limb				62	13	75	25
Cartilage	23	98	31				
Control hind limb				62	12	74	26
Immobilized hind limb				59	10	69	31
Cartilage	28	78	30				
Control hind limb				52	18	70	30
Immobilized hind limb				50	17	67	33
Cartilage	29	82	34				
Control hind limb				55	17	71	29
Immobilized hind limb				53	17	70	30
Cartilage	30	85	37				
Control hind limb				55	16	71	29
Immobilized hind limb				54	15	69	31
Cartilage	31	89	41				
Control hind limb				52	15	67	33
Immobilized hind limb				47	12	59	41
Cartilage	11	87	28				
Control hind limb				57	15	72	28
Immobilized hind limb				54	15	69	31
Group II: 99–124 days of age, 33–58 days of immobilization							
Cartilage	10	99	49				
Control hind limb				55	15	70	30
Immobilized hind limb				55	12	67	33
Cartilage	24	102	33				
Control hind limb				60	10	70	30
Immobilized hind limb				65	8	73	27
Cartilage	25	108	38				
Control hind limb				63	12	75	25
Immobilized hind limb				66	10	76	24
Cartilage	26	111	44				
Control hind limb				66	11	77	23
Immobilized hind limb				64	11	75	25
Cartilage	20	117	47				
Control hind limb				57	10	67	33
Immobilized hind limb				59	11	70	30
Cartilage	12	118	58				
Control hind limb				61	10	71	29
Immobilized hind limb				55	11	66	34
Cartilage	21	124	47				
Control hind limb				65	10	75	25
Immobilized hind limb				58	10	68	32

* Small subscript $_s$ refers to 100 grams of cartilage solids.
(F)$_s$ = grams of connective-tissue solids in 100 grams of cartilage solids.
CSA$_s$ = weight of chondroitin sulphate calculated from Levene's molecular weight.
(E)$_s$ = grams of extracellular solids in 100 grams of cartilage solids = (F)s + CSAs.
(C)$_s$ = grams of intracellular solids in 100 grams of cartilage solids = 100 − (E)s.

TABLE III *

PHASE MASS DATA FOR ONE KILOGRAM OF ARTICULAR CARTILAGE

Derived Solids

	Dog (*No.*)	Age (*Days*)	Immobilized (*Days*)	Total Water (*gm.*)	Total Solids (*gm.*)	$(F)_S$ (*gm.*)	CSA_S (*gm.*)	$(C)_S$ (*gm.*)
Group I: 78–98 days of age. 28–41 days of immobilization								
Cartilage	22	95	28					
Control hind limb				786	214	136	28	50
Immobilized hind limb				808	192	119	25	48
Cartilage	23	98	31					
Control hind limb				789	211	135	24	52
Immobilized hind limb				815	185	109	18	58
Cartilage	28	78	30					
Control hind limb				807	193	101	35	57
Immobilized hind limb				816	184	92	31	61
Cartilage	29	82	34					
Control hind limb				798	202	112	34	56
Immobilized hind limb				813	187	100	32	55
Cartilage	30	85	37					
Control hind limb				800	200	111	32	57
Immobilized hind limb				808	192	103	29	60
Cartilage	31	89	41					
Control hind limb				801	199	104	30	65
Immobilized hind limb				828	172	81	21	70
Cartilage	11	87	28					
Control hind limb				794	206	118	31	57
Immobilized hind limb				816	184	99	27	58
Group II: 99–124 days of age. 33–58 days of immobilization								
Cartilage	10	99	49					
Control hind limb				788	212	116	32	63
Immobilized hind limb				795	205	113	25	67
Cartilage	24	102	33					
Control hind limb				791	209	125	21	63
Immobilized hind limb				827	173	110	15	48
Cartilage	25	108	38					
Control hind limb				780	220	138	27	55
Immobilized hind limb				801	199	130	20	49
Cartilage	26	111	44					
Control hind limb				781	219	144	25	50
Immobilized hind limb				813	187	119	21	47
Cartilage	20	117	47					
Control hind limb				790	210	119	21	69
Immobilized hind limb				821	179	106	19	54
Cartilage	12	118	58					
Control hind limb				791	209	128	22	59
Immobilized hind limb				815	185	101	20	64
Cartilage	21	124	47					
Control hind limb				776	224	145	24	55
Immobilized hind limb				843	157	90	15	52

* Units are expressed per kilogram of fresh cartilage as indicated by subscript of capital s.

All estimated values for $(F)_S$; CSA_S; $(E)_S$; and $(C)_S$ are obtained from the weights in 100 grams of cartilage solids in Table II and the determined total solids.

$(F)_S$ = grams connective-tissue solids per kilogram fresh cartilage = $(F)_s \times$ total solids/100.

CSA_S = grams chondroitin sulphate per kilogram fresh cartilage = $CSA_s \times$ total solids/100.

$(E)_S$ = extracellular solids per kilogram fresh cartilage = $(F)_S + CSA_S$.

$(C)_S$ = intracellular solids per kilogram fresh cartilage = total determined solids $- (E)_S$.

TABLE IV *

PHASE MASS DATA FOR ONE KILOGRAM CARTILAGE

Derived Water

	$(F)_S$ (gm.)	$(H_2O)_F$ (gm.)	$(Cl)_F$ (mEq.)	$(Cl)_S$ (mEq.)	Cl_S (mEq.)	Cl_U (mEq.)	$(H_2O)_U$ (gm.)	$(H_2O)_E$ (gm.)	$(E)_S$ (gm.)	$(H_2O)_C$ (gm.)	$(C)_S$ (gm.)	$(H_2O)_C$ (gm.)
Group I												
Dog 22 Cartilage												
Control	136	213	28.1	68.2	40.1	122.1	328	541	164	245	50	830
Immobilized	119	188	24.6	73.8	49.2		403	591	144	217	48	819
Dog 23 Cartilage												
Control	135	212	27.9	72.1	44.2	125.8	350	562	157	225	54	806
Immobilized	109	171	22.5	74.6	52.1		414	585	127	230	58	799
Dog 28 Cartilage												
Control	101	158	20.8	66.0	45.2	124.6	363	521	136	285	58	831
Immobilized	92	144	19.1	69.1	50.1		402	546	123	270	61	816
Dog 29 Cartilage												
Control	112	176	23.1	69.6	46.5	123.1	378	554	146	244	56	813
Immobilized	100	157	20.6	65.8	45.2		367	524	132	289	55	840
Dog 30 Cartilage												
Control	111	174	22.9	58.8	35.9	125.1	287	461	143	339	57	856
Immobilized	103	162	21.7	65.3	44.0		352	514	132	294	60	840
Dog 31 Cartilage												
Control	104	163	21.5	72.5	51.0	125.7	406	569	134	232	65	781
Immobilized	81	127	16.7	68.5	51.7		412	539	102	289	70	805
Dog 11 Cartilage												
Control	118	185	24.4	70.3	45.9	123.1	402	587	149	207	57	784
Immobilized	99	155	20.4	75.2	54.8		445	600	126	216	58	788
Group II												
Dog 10 Cartilage												
Control	116	182	23.9	68.7	44.8	129.4	346	528	148	261	63	805
Immobilized	113	177	23.1	64.8	41.7		322	499	138	296	67	810
Dog 24 Cartilage												
Control	125	196	25.8	69.9	44.1	125.2	352	548	146	243	63	802
Immobilized	110	173	22.7	79.6	56.9		454	627	125	200	48	806
Dog 25 Cartilage												
Control	138	217	28.5	68.0	39.5	123.2	321	538	165	242	55	815
Immobilized	130	204	26.8	76.0	49.2		399	603	150	198	49	805
Dog 26 Cartilage												
Control	144	226	29.7	67.8	38.1	122.0	312	538	162	243	50	830
Immobilized	119	187	24.6	65.0	40.4		331	518	140	295	47	862
Dog 20 Cartilage												
Control	119	187	24.6	69.6	45.0	131.0	343	530	140	261	69	791
Immobilized	106	166	21.9	73.7	51.8		395	561	125	260	54	828
Dog 12 Cartilage												
Control	128	201	26.4	72.2	45.6	127.5	359	560	150	231	59	797
Immobilized	101	158	20.8	80.7	59.8		469	627	121	188	64	746
Dog 21 Cartilage												
Control	145	228	29.9	66.4	36.5	123.0	297	525	169	251	55	820
Immobilized	90	141	18.6	67.3	48.7		396	537	105	300	52	857

*$(F)_S$ = Table III.
$(H_2O)_F$ = grams water associated with $(F)_S = 157 \times (F)_S/100$.
$(Cl)_F$ = milliequivalents chloride associated with $(F)_S = 20.65 \times (F\ s/100$.
$(Cl)_S$ = milliequivalents chloride per kilogram fresh cartilage = $(Cl)_s \times$ total solids/100.
$Cl_S = (Cl)_S - (Cl)_F$.
Cl_U = milliequivalents chloride per kilogram serum water $\times$ 0.0099/0.0095.
$(H_2O)_U$ = grams ultrafiltrate water = $Cl_S \times 1000/Cl_U$.
$(H_2O)_E = (H_2O)_F + (H_2O)_U$.
$(E)_S$ = see Table III = $(F)_S$ + CSAs.
$(H_2O)_C$ = total water − $(H_2O)_E$.
$(C)_S$ = see Table III = total determined solids − $(E)_S$.
$(H_2O)_C$ = grams of water per kilogram of chondrocytes = $(H_2O)_C \times 1000/(H_2O)_C + (C)_S$.

mass the remainder of the 100 grams. It will be noted that the mass of the extracellular solids decreased in the cartilage from the immobilized extremity as the result of a decrease in both the connective-tissue fibers and the chondroitin sulphate. These data are shown graphically in Chart IV.

When one considers a kilogram of fresh cartilage, the mass of the extracellular compartment consisting of fluids and solids, and the mass of the intracellular compartment (chondrocytes), consisting of solids and water, both have to be derived. Table III gives the derived mass of the extracellular solids $(E)_s$ and the derived mass of the chondrocyte solids $(C)_s$. Table IV gives the derived fluid or water values per kilogram of fresh cartilage. It is informative to divide the extracellular fluids into water associated with the connective-tissue fibers

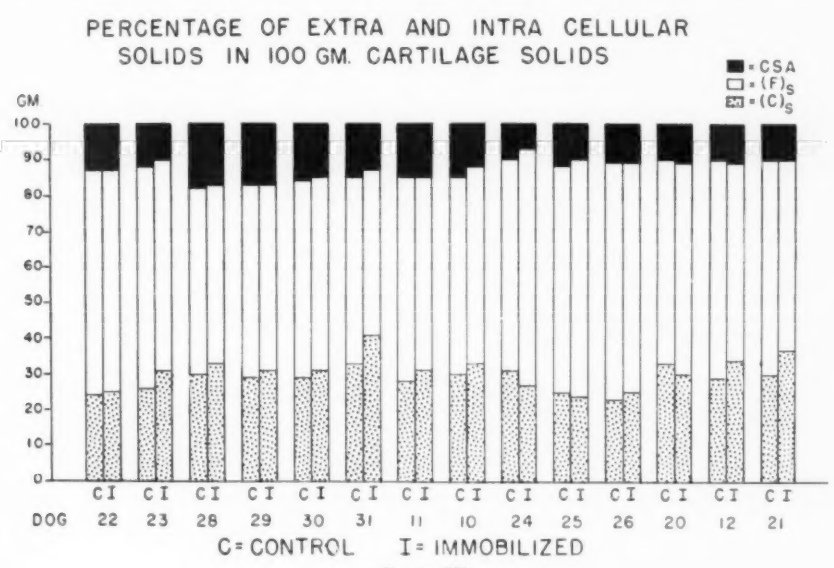
PERCENTAGE OF EXTRA AND INTRA CELLULAR SOLIDS IN 100 GM. CARTILAGE SOLIDS
■ = CSA
□ = $(F)_s$
▨ = $(C)_s$
GM
100
90
80
70
60
50
40
30
20
10
0
C I C I C I C I C I C I C I C I C I C I C I C I C I C I
DOG 22 23 28 29 30 31 11 10 24 25 26 20 12 21
C = CONTROL I = IMMOBILIZED

CHART IV

Graphic representation of the percentage of extracellular and intracellular solids in 100 grams of total cartilage solids. The percentage of the chondroitin sulphate solids illustrated by the black areas, and the percentage of the connective-tissue solids illustrated by the unshaded areas, represent the extracellular solids. The percentage of the chondrocyte solids is represented by the dotted areas.

$(H_2O)_F$ and the ultrafiltrate water $(H_2O)_U$, the sum of which makes up the total water of the extracellular compartment $(H_2O)_E$. The derived extracellular water was subtracted from the total determined water to obtain the chondrocyte water in a kilogram of fresh cartilage.

Graphic representations of the patterns for each kilogram of fresh cartilage are given in Charts V and VI. Each pair of columns illustrates cartilage values from one animal. The first column represents a kilogram of control cartilage and the adjoining column the cartilage from an immobilized limb. The major points of interest in these patterns are:

1. The extracellular solids in every immobilized cartilage decreased at the expense of both the connective-tissue-fiber solids and the chondroitin sulphate.

2. The extracellular water increased in volume. This increase was the result of an increase in the ultrafiltrate volume because the water associated with the connective-tissue fibers actually decreased in volume. It is a possibility that the decrease in the connective-tissue fibers and in the chondroitin sulphate could result from the diluting effect of the increased volume of the ultrafiltrate.

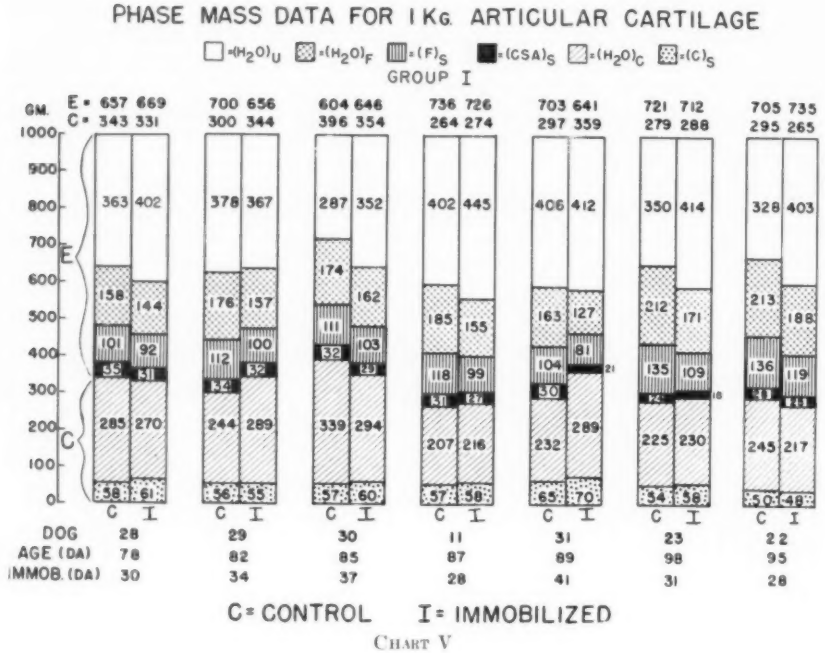

CHART V

3. While the absolute value for water content of the chondrocytes $(H_2O)_c$ was somewhat erratic, the percentage of the intracellular water, when the control value is compared with the experimental value, was quite constant. For example, in Dog 22, the percentage of water in the control chondrocytes was 83; in the experimental chondrocytes the percentage was 82.

DISCUSSION

In a group of puppies such as these, chemical changes take place that are the result of age differences [5], and there are also changes that take place as a result of the immobilization of the extremity. In this paper the discussion will be confined to the changes resulting from the atrophy following immobilization. For this reason the cartilage from the control limb must be compared with the cartilage from the immobilized limb of each individual puppy in order to rule out the age effects.

The volume increase in the total water content of the cartilage (Chart I) is an important fact. However, showing that this increase in water is the result of an increase in the ultrafiltrate volume is still more important. This deduction is the same in articular cartilage as was found in the skeletal muscle [7] from the same limb. It appeared also in experimental atrophy following denervation [1]. Therefore, whether the atrophy resulted from experimental denervation or from experimental immobilization of an extremity, the extracellular fluid volume in cartilage increased. Since this increased extracellular water volume is common to both forms of experimental atrophy, it implies that disuse must be the responsible factor in the change.

Further changes in the composition of the extracellular compartment, when comparisons of the control and immobilized cartilage are made, were the decreases in the amounts of both connective tissue and chondroitin sulphate. It will be noted from Tables II and III and from Charts II and III that the percentage decrease in each of these two constituents was not as large when 100 grams of cartilage

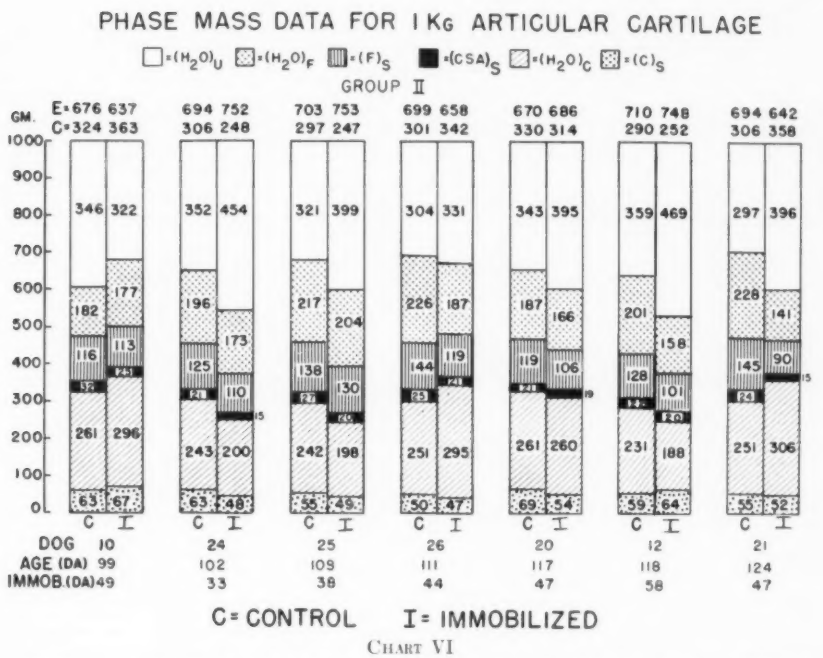

CHART VI

Charts V and VI: Phase mass data for one kilogram of fresh cartilage. The first column of each pair presents the data from the weight-bearing control limb, labeled C; and the adjoining column presents the data from the immobilized limb, labeled I. Graphic representation of the extracellular phase consisting of (1) the ultrafiltrate water illustrated by the clear areas, (2) the connective-tissue phase consisting of the connective-tissue solids illustrated by the perpendicular cross-hatched areas and the associated water illustrated by the double cross-hatched areas, and (3) the chondroitin sulphate phase illustrated by the black area. The intracellular phase consisting of the intracellular water illustrated by the single diagonally hatched areas and the intracellular solids illustrated by the dotted areas.

solids were considered as when a kilogram of the fresh tissue was considered. This suggests that the low determined values of these two constituents were the result of dilution of the extracellular compartment in the experimental cartilage. This finding is different from that observed in atrophy following denervation where the mass of chondroitin sulphate decreased per 100 grams of cartilage solids as well as per kilogram of fresh cartilage. These results imply that in disuse atrophy alone the chondroitin sulphate decreased in concentration while in atrophy from denervation there was a decrease in concentration as well as in formation or production or breakdown.

The relationship of sodium to chondroitin sulphate and connective tissue in cartilage is vital and is given in Table V. The total determined sodium values per kilogram of fresh cartilage $(Na)_T$ are given in the second column of figures. These values are corrected for the sodium associated with the connective-tissue fibers and the sodium in the ultrafiltrate volume, leaving an amount of sodium that is labeled $\triangle$Na. These partitions of sodium are shown graphically in Chart VII. This $\triangle$Na must be covering some of the chondroitin sulphuric acid, making a sodium binding compound [12]. Funcoka was unable to demonstrate any sodium in cartilage cells. It will be noted from Chart VIII that the percentage change in the $\triangle$Na runs parallel to that of the chondroitin sulphate. Thus when chondroitin sulphate decreased, the $\triangle$Na also decreased. Referring again to Charts II and III in which percentage change in total sodium is shown per 100 grams of cartilage solids and also per kilogram of fresh cartilage, it will be seen that the only low percentage decreases are in the fresh cartilage values and not in the solid values.

TABLE V *

EFFECT OF IMMOBILIZATION ON THE SODIUM AND CHONDROITIN SULPHATE VALUES IN CARTILAGE

Tissue	$[Na]_F$ (*mEq.*)	Extracellular Phase: Total Na $(Na)_T$ (*mEq.*)	Extracellular Phase: [Na associated with $(F)_S$ (*mEq.*) + Na in $(H_2O)_U$] (*mEq.*)	ΔNa (*mEq.*)	CSA (*mM.*)
Dog 22					
Serum	150.3				
Cartilage					
Control		188.4	27.4	49.3	111.7	60
Immobilized		170.7	24.0	60.6	86.1	53
Dog 23					
Serum	153.2				
Cartilage					
Control		204.0	27.3	53.9	122.8	51
Immobilized		179.8	22.0	63.4	94.4	38
Dog 28					
Serum	149.1				
Cartilage					
Control		252.5	20.4	54.1	178.0	75
Immobilized		230.1	18.6	59.9	151.6	66
Dog 29					
Serum	148.6				
Cartilage					
Control		242.2	22.6	56.2	163.4	73
Immobilized		224.9	20.2	54.5	150.2	68
Dog 30					
Serum	144.5				
Cartilage					
Control		192.2	22.4	41.5	128.3	68
Immobilized		185.2	20.8	50.8	113.5	62
Dog 31					
Serum	149.9				
Cartilage					
Control		213.5	21.0	60.8	131.7	64
Immobilized		195.3	16.5	61.7	117.2	45
Dog 11					
Serum	147.9				
Cartilage					
Control		211.1	23.8	59.4	127.8	66
Immobilized		193.7	20.0	65.8	107.9	58
Dog 10					
Serum	149.5				
Cartilage					
Control		192.4	23.4	51.7	117.2	68
Immobilized		193.3	22.8	48.1	122.3	53
Dog 24					
Serum	147.0				
Cartilage					
Control		180.0	25.2	51.7	103.1	45
Immobilized		170.5	22.2	66.7	81.6	32
Dog 26					
Serum	147.2				
Cartilage					
Control		172.0	29.1	44.7	98.2	53
Immobilized		159.3	24.0	48.7	86.6	45
Dog 12					
Serum	151.0				
Cartilage					
Control		191.9	25.8	54.2	111.9	47
Immobilized		98.0	20.0	70.8	97.4	43

* $[Na]_F$ = milliequivalents Na per kilogram extracellular fluid.
$(Na)_T$ = milliequivalents Na per kilogram fresh cartilage.
In $(H_2O)_U$ = milliequivalents Na in ultrafiltrate volume.
Associated with $(F)_S$ = milliequivalents Na associated with $(F)_S = 20.2 \times (F)_S/100$.
ΔNa = total determined sodium − Na in the extracellular phase.
CSA = mM. chondroitin sulphate per kilogram of fresh cartilage.

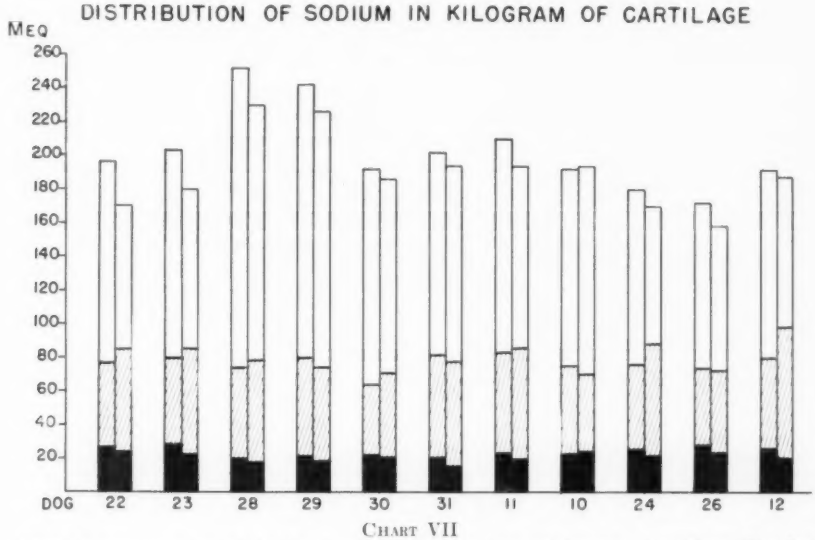

CHART VII

Graphic representation of the distribution of sodium in one kilogram of cartilage. The first column of each pair represents the total amount of sodium per kilogram of cartilage from the control limb, and the adjoining column represents the total sodium content from the immobilized limb. The milliequivalents of sodium associated with the weight of the connective-tissue solids or the fiber solids $(F)_s$ per kilogram of cartilage are illustrated by the black areas; the milliequivalents of sodium in the ultrafiltrate $(H_2O)_c$ of the extracellular compartment illustrated by the single diagonally hatched areas, and the remainder of the sodium called $\triangle Na$, illustrated by the clear areas.

The low values in the fresh cartilage are mostly the result of a lowered sodium concentration covering the low chondroitin sulphate concentrations. It seems, therefore, that these relationships of sodium and chondroitin sulphate values in the fresh cartilage following immobilization are the result of dilution by the ultrafiltrate volume rather than some other cause.

It is well known that the environmental fluids around the cells of a tissue are of paramount importance to the integrity of the cells, their size, water content, and metabolism. In a tissue such as cartilage, as distinguished from the soft tissues, the environment of the cells consists not only of fluids but also of connective tissue and chondroitin sulphate. The integrity of the chondrocytes, their size, water content, and metabolism have many variables. In the types of atrophy studied by us the changes found in the environment of the chondrocytes require consideration of these cells.

In atrophy of articular cartilage following denervation, the extracellular fluids increased in volume significantly while the chondroitin sulphate decreased in amount as a result of not only dilution but also of either non-production or disintegration. Associated with this change in their extracellular environment, the chondrocytes were found to have a low percentage of cell water, signifying a disturbance in their cytoplasm. Whether this disturbance in the cytoplasm of the chondrocytes diminished the production of the chondroitin sulphate or the disintegration of the chondroitin sulphate caused a disturbance in the integrity of the chondrocytes is not known at this time.

In the present study of disuse atrophy following immobilization it has been shown that the extracellular fluids of the cartilage also increased in volume and the chondroitin sulphate and connective-tissue masses also decreased, but only as a result of dilution and not of non-production or breakdown. In Table IV are listed the grams of water per kilogram of chondrocytes $\{H_2O\}_c$, first of the control and then of the experimental cartilage. Unlike the chondrocytes in atrophy from

TABLE VI*

DISTRIBUTION OF POTASSIUM BETWEEN SERUM AND CARTILAGE PHASES

Tissue	$[K]_F$ (*mEq.*)	Total K $(K)_T$ (*mEq.*)	In Extracellular Phase: In $(H_2O)_U$ + Associated with $(F)_S$ (*mEq.*)	In (C) (*mEq.*)	$(K)_C$ (*mEq.*)	$[K]_C$ (*mEq.*)
Dog 22						
Serum	5.49					
Cartilage						
Control		24.9	1.8	3.0	20.1	68.1	82.0
Immobilized		22.1	2.2	2.6	17.3	65.3	79.7
Dog 23						
Serum	5.16					
Cartilage						
Control		24.0	1.8	3.0	19.2	68.8	85.3
Immobilized		24.0	2.1	2.4	19.5	67.7	84.8
Dog 28						
Serum	5.05					
Cartilage						
Control		25.8	1.8	2.2	21.7	63.3	76.1
Immobilized		24.3	2.0	2.0	20.2	61.6	74.9
Dog 29						
Serum	5.33					
Cartilage						
Control		24.2	2.0	2.5	19.7	65.6	80.7
Immobilized		21.9	1.9	2.3	17.8	51.7	61.6
Dog 30						
Serum	5.12					
Cartilage						
Control		22.2	1.5	2.4	18.3	46.2	53.9
Immobilized		23.9	1.8	2.3	19.8	55.9	67.4
Dog 31						
Serum	5.08					
Cartilage						
Control		19.7	2.1	2.3	15.4	51.8	66.3
Immobilized		20.6	2.1	1.8	16.7	46.5	57.8
Dog 11						
Serum	4.95					
Cartilage						
Control		22.3	2.0	2.6	17.7	67.1	85.5
Immobilized		23.5	2.2	2.2	19.1	69.8	88.6
Dog 10						
Serum	4.74					
Cartilage						
Control		26.1	1.6	2.5	21.9	67.7	84.1
Immobilized		23.7	1.5	2.5	19.7	54.0	84.1
Dog 24						
Serum	5.10					
Cartilage						
Control		21.4	1.8	2.7	16.9	55.8	69.5
Immobilized		19.9	2.3	2.4	15.2	61.3	76.0
Dog 26						
Serum	4.93					
Cartilage						
Control		23.2	1.5	3.2	18.5	61.5	73.7
Immobilized		19.0	1.6	2.6	14.7	43.1	50.0
Dog 12						
Serum	4.73					
Cartilage						
Control		23.1	1.7	2.8	18.6	64.0	80.3
Immobilized		20.3	2.2	2.2	15.9	63.0	85.5

*$[K]_F$ = milliequivalents K per kilogram of extracellular fluid.
$(K)_T$ = milliequivalents K per kilogram of fresh cartilage.
In $(H_2O)_U$ = milliequivalents K in ultrafiltrate volume.
Associated with $(F)_S$ = milliequivalents K associated with $(F)_S = 2.2 \times (F)_S/100$.
In (C) = milliequivalents K in chondrocytes = $(K)_T$ − (in $(H_2O)_U$ + associated with $(F)_S$).
$(K)_C$ = milliequivalents K per kilogram of chondrocytes.
$[K]_C$ = milliequivalents K per kilogram of chondrocyte water.

denervation, the percentage of intracellular water in the chondrocytes from the immobilized cartilage had not changed in twelve of the fourteen pairs of cartilage presented, indicating that the functioning cytoplasm of the chondrocytes had not changed.

Furthermore, in eight of the eleven pairs of cartilage in which there was enough material for the analysis of potassium, approximately the same concentration of potassium was found in each pair when expressed per kilogram of chondrocytes $\{K\}_c$ or per kilogram of chondrocyte water $[K]_c$ (Table VI). These values for potassium suggest that the chondrocytes following immobilization atrophy have maintained their intracellular concentration of this cation.

It seems, therefore, that the change in the production or the depletion by disintegration of the chondroitin sulphate was caused by a disturbance in the integrity of the chondrocytes since in both forms of experimental atrophy there was an increase in the extracellular fluid volume and a resultant decrease in the con-

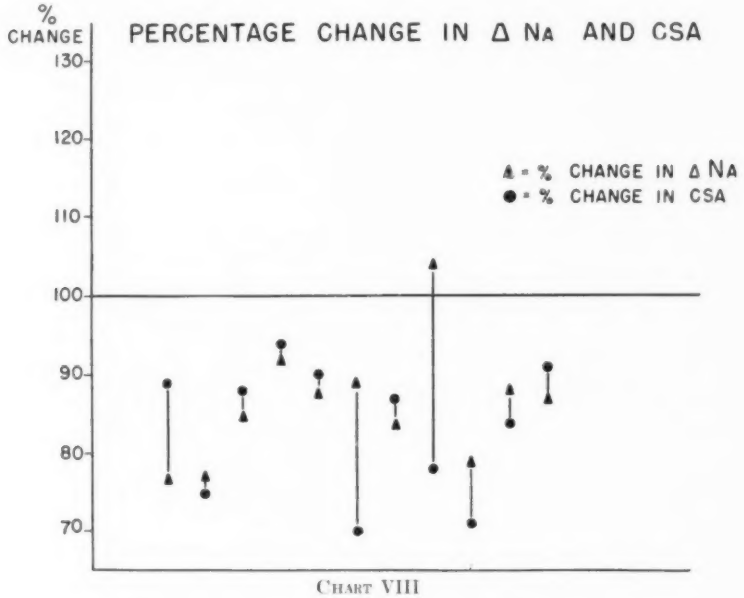

CHART VIII

centration of chondroitin sulphate by dilution. Only when the chondroitin sulphate decreased beyond that attributable to dilution were there changes in the integrity of the chondrocytes. This substantiates the assumption made by Sylven that "chondroitin sulphate occupies the central position in cartilage biology, and probably plays a very important role in the metabolism and function of cartilage". It seems probable therefore that the reversibility of atrophy in articular cartilage depends upon the integrity of the chondrocytes which in turn depends upon numerous factors, one of which is apparently chondroitin sulphate production. These findings should help clarify the problem of when to expect atrophy in cartilage to be reversible.

SUMMARY

By means of histochemical procedures the characterization of the compartments of articular cartilage, the distribution of water, electrolytes, connective tissue, and chondroitin sulphate in these compartments during immobilization atro-

phy have been described. The results obtained support the following conclusions concerning the cartilage during immobilization of an extremity:

1. The total water content increased as the result of an increase in the ultrafiltrate volume in the extracellular compartment.

2. The amounts of both the connective tissue and the chondroitin sulphate in fresh cartilage decreased but these percentage decreases were not so large as when 100 grams of cartilage solids were considered, suggesting that the low values of these two constituents were the result of dilution of the extracellular compartment with the increased ultrafiltrate volume.

3. The $\triangle$Na values (total determined sodium minus the sodium in the extracellular phase) were low paralleling the low chondroitin sulphate values and thus giving further support to the conclusion that the low chondroitin sulphate values were the result of dilution.

4. In the intracellular compartment of the chondrocytes, the percentage of intracellular water did not change; nor was there any change in the concentration of potassium which is the cation present in the chondrocyte indicating that the functioning cytoplasm of the chondrocytes had not altered during immobilization atrophy.

5. Considering the two types of experimental atrophy in cartilage from denervation reported previously and disuse reported here, our chemical data and their interpretations have afforded a basis for some deductions on when atrophy is reversible, as in disuse, and when it is not reversible, as in atrophy following denervation.

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The Vascular Response to Fracture of the Tibia in the Rat*†

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The existence of a hyperemic response to fracture has been assumed since the pioneer work of Lexer in the early years of the twentieth century. Lexer [9] and his pupil Rodhe [10] injected the arterial system of freshly severed limbs of experimental animals with a mixture of turpentine and mercury. They then made roentgenograms of the part and were able to show considerable visual evidence of an increase in the number and size of vessels in the region of a fracture. Somewhat later Kolodny [6] utilized a similar technique. Although his studies were less extensive than those of Lexer he also concluded that a local hyperemia follows fracture. The subject has received very little experimental attention since.

The arteriographic techniques that were available to Lexer and Kolodny possessed serious limitations and could hardly be considered proof acceptable to modern scientific standards. There are obvious difficulties in attempting a quantitative evaluation of a three-dimensional vessel system with a two-dimensional roentgenogram. In addition, it is doubtful if the authors were ever able to visualize vessels of arteriolar or smaller size.

With the development of modern plastic-injection techniques by Batson[1], it became possible to produce an accurate mold of the circulatory system of a part. After removal of the surrounding soft tissue by maceration, a three-dimensional model remains that will allow critical study. Knisely [5] has recently shown that a very large proportion of the vascular tree down to and including capillaries is demonstrated by this technique. In the present study, we have utilized the basic principles of Batson's work to test the validity of Lexer's observation that the local vascular system enlarges after fracture. All data obtained were subjected to statistical analysis. The purpose of this paper is to present the results of this work and to discuss the significance of the findings.

MATERIALS AND METHODS

Four-month-old Wistar strain male albino rats were used as the experimental animal. The animals were fed a standard laboratory chow and were housed in cages, fourteen by eighteen inches, in groups of ten.

A closed fracture of the left tibia and fibula was produced by finger manipulation with the animal under ether anesthesia. The fractures were not immobilized, and the animals were allowed to run free in their cages. All animals were discarded who exhibited a break in the skin of the fractured limb at any time during the experiment. A group of normal uninjured animals were used as controls. At designated times after fracture the animals were killed, and the lower trunk and hind limbs injected with a plastic solution. Techniques were identical in fracture and control animals.

At death the animals were weighed, the sternum was resected, and the thoracic aorta cannulated with a saline-filled No. 18 polyethylene tube attached proximally to a three-way stopcock and saline-filled fifty-cubic-centimeter syringe. A stab

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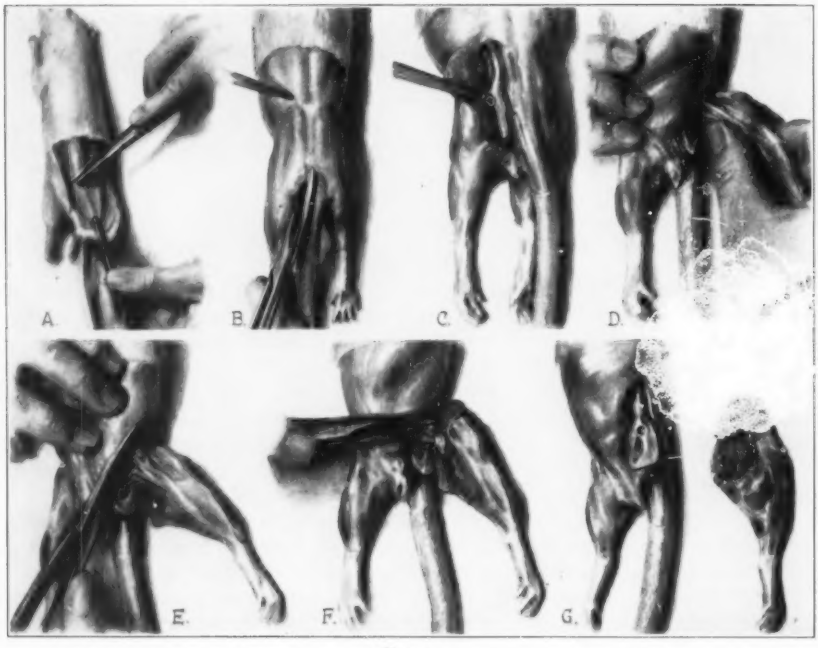

FIG. 1

Illustration showing the technique used in disarticulation of all limbs used in the experiment: *A*, the cadaver is skinned; *B*, the fascial attachment of the gluteus maximus to the spine is cut; *C*, the abductors have been reflected from the ilium by thumb-nail dissection and the hamstrings dissected from their origin on the ischial tuberosity; *D*, the animal is turned on its back, and the adductors detached cleanly from the ischium and pubic rami with the thumb nail; *E*, the iliopsoas muscle and great vessels of the limb are sectioned at the level of the inguinal ligament with sharp scissors; *F*, the capsule and short rotators of the hip joint are cut; and *G*, following disarticulation the innominate bone is bare and all muscle has been reflected with the limb.

wound was made in the right ventricle. The vascular tree of the trunk and lower extremities was then flushed with approximately forty cubic centimeters of normal saline solution. Toward the end of this injection, a twenty-cubic-centimeter syringe, filled with liquid methyl methacrylate mixed with promoter and catalyst was attached to the system to permit a continuous flow of liquid. Care was taken to avoid air bubbles in the system. A complete injection required about ten cubic centimeters of plastic, at the end of which the hind limbs were rigidly extended and the plastic was flowing freely from the puncture wound in the right ventricle. The pressure of injection was not measured, but required strong hand power. All animals in which technical problems arose during injection, such as slipping of the cannula and bubbles in the system, were discarded from the series. The animals were refrigerated for forty-eight hours to assure complete hardening of the plastic.

The hind limbs of each animal were then disarticulated at the hip (Fig. 1). All disarticulations were done by the same worker, who used a constant technique. After the lower portion of the animal had been skinned, the cadaver was placed on its abdomen. Scissors were used to release the gluteus maximus at its fascial attachment to the spine, and the abductor muscles were peeled from the ilium by thumb-nail dissection. The hamstrings were then detached at their origins on the ischial tuberosity by use of a pair of sharp dissection scissors applied closely to the bone. The cadaver was then turned on its back, and the adductor group of muscles were detached by thumb-nail dissection. The iliopsoas was sharply sec-

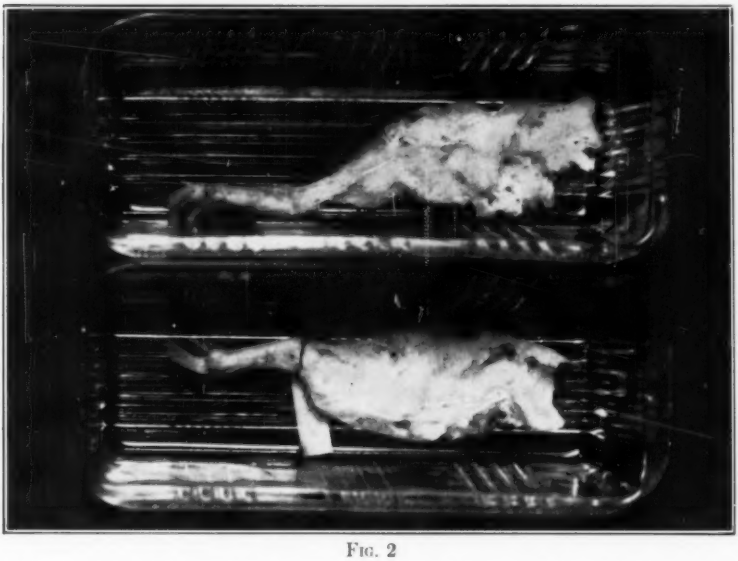

FIG. 2

Paired limbs nine days after fracture are shown following chemical digestion with potassium hydroxide and hydrochloric solutions. They will then be dried and weighed. Note that the labeled limb, which is the left, appears larger than the right.

tioned at the level of the inguinal ligament to expose the capsule of the hip joint. Both capsule and rotatory muscles were cut to complete the disarticulation. The left limb was labeled immediately and paired with the opposite member. Roentgenograms were then made of the fractured left limbs. All specimens not exhibiting an obviously complete fracture were discarded. Each limb was then treated in a small glass container that was labeled for identification. All soft tissue was removed by immersion in a 20 per cent solution of potassium hydroxide for seventy-two hours. The specimen was then gently flushed with tap water and soaked in a 30 per cent hydrochloric acid solution for forty-eight hours to remove the remaining mineral content of bone.

At the completion of chemical digestion all material had been removed, leaving a plastic mold of the vascular tree of the limb (Fig. 2). Specimens were dried at room temperature for forty-eight hours and were then weighed with an analytical balance scale*.

Records were kept of the weight of the rat, weights of the plastic casts of right and left limbs, and the time interval from fracture until death of the animal.

RESULTS

One hundred and fifty-nine animals were evaluated according to the accumulated data (Table I). Twenty-five control animals were used. Fourteen of the control animals were injected at the beginning, and eleven were injected near the end of the experiment. Limb weights of both groups were combined after it was established that their mean differences were not of significance. Throughout the experiment the weight of the left limb was subtracted from that of the right. A plus value thus indicates that the right limb was heavier, and a minus value that the left limb was heavier.

The mean difference in limb weights for each group of rats was compared

* Model AB-4 Christian Becker notched beam chainomatic dial reading analytical balance scales.

TABLE I*

DATA OBTAINED FROM THE EVALUATION OF 159 RATS

Days after Fracture	Number of Rats	Mean Difference $\bar{X}_D$ gm.	P(XD) (compared c zero)	$\bar{X}_D$(control) — $\bar{X}_D$ exp.	P
Control	25	+ 0.041	<0.3 >0.2	———	———
3	17	+ 0.010	<0.7 >0.5	+ 0.031	<0.5 >0.3
6	19	− 0.005	<0.9 >0.7	+ 0.046	<0.3 >0.2
9	17	− 0.087	<0.01>0.001	+ 0.128	<0.01>0.001
12	15	− 0.063	<0.2 >0.1	+ 0.104	<0.1 >0.05
15	17	− 0.036	<0.2 >0.1	+ 0.077	<0.1 >0.05
18	16	+ 0.005	<0.9 >0.7	+ 0.036	<0.5 >0.4
27	16	− 0.014	<0.5 >0.3	+ 0.053	<0.3 >0.2
36	17	+ 0.024	<0.3 >0.2	+ 0.017	<0.7 >0.5

* Pertinent data on 159 rats are shown here. Column $\bar{X}D$ lists the mean difference between right and left limbs of each group. A plus value indicates that the right limb was heavier, and a negative value that the left limb was heavier. The values of t are given in their respective columns. In column P ($\bar{X}D$) the mean difference has been compared with zero to test for significance. In column $\bar{X}D$ (control) $-\bar{X}D$ exp. the mean differences of the experimental animals are subtracted from those of the controls. The probability that these observations might occur by chance is given in Column P.

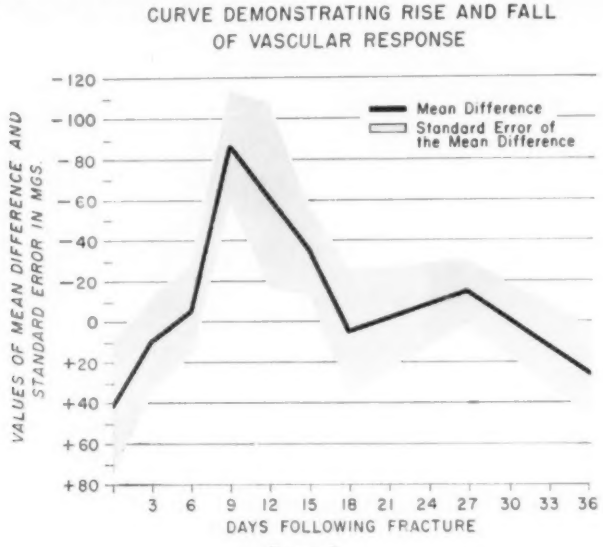

CHART I

Curve showing the rise and fall of the vascular response following fracture of the tibia in the rat. The values of the mean differences in milligrams are plotted against the number of days following fracture and are seen as a solid black line. The stippled band represents the range of the standard error of the mean differences.

against zero and against the mean difference of the control group to demonstrate the presence or absence of significant variation in values. The values of t for each group are shown and the probabilities that these differences might occur by chance are given in the columns marked *P(XD)* and *P*, respectively.

A close examination of the values of the mean differences reveals that they are undergoing a gradual shift to negative values at three and six days. At nine days after fracture the mean difference has reached a negative value of statistical significance.

After nine days the negative values of the mean differences gradually decrease to assume a positive value at thirty-six days.

When the values of the mean differences in milligrams and the standard error of the mean differences are plotted a definite pattern in the response can be seen. In Chart I the value of the mean differences is represented by a single line, whereas the standard error is plotted as a stippled band.

Both values indicate that the vascular volume of the fractured limb begins to increase rapidly by the third day. This increase reaches a peak at the ninth day and is then followed by a gradual decline to approximate control values at thirty-six days. The dip in the curve seen at eighteen days is probably within the range of experimental error and arises from a rather marked difference seen in two animals of the group. A scientifically acceptable method of determining fracture union was not employed in this experiment, but, on the basis of roentgenographic and clinical evidence, reasonably firm union appeared to have occurred by the twenty-seventh day.

DISCUSSION

A measurable enlargement of the local vascular bed of the rat occurs after a closed fracture of the tibia. This process reaches a peak at nine days and then gradually subsides to control levels with union of the fracture. Other writers have used the terms *hyperemia* [9,10] or *inflammation* [7] in describing the vascular phenomena associated with fracture. The changes observed in this study might more exactly be described by the term *hyperplasia* to distinguish them from the transitory hyperemia seen after the application of external heat to a part. They would seem to arise from the fixed enlargement of existing vessels and from the formation of new channels by capillary budding. Although we have made no specific effort to localize the origin of the enlarging vascular system, preliminary observations suggest that the vessels of the periosteum and surrounding soft tissues play the predominant role. On the other hand, the ability of the capillary bed to proliferate has been well documented by others [11], and almost all writers [3,8,12] on the subject of fracture histology recognize marked capillary proliferation in the fracture callus.

In rats with fractures identical to those in this study [2], it was noted that cellular proliferation in the periosteum reached a peak at ten to twelve days after fracture. This closely approximates the peak of the vascular response that we have demonstrated and suggests that a coordinated effort between vascular proliferation and osteogenic-cell proliferation occurs. In this regard several workers [4,12] have noted primary bone in close approximation to capillary buds.

Certain limitations have been encountered in the technique used in this study. Complete filling of the capillary bed is probably not feasible. Knisely [5] suggests that submaximal filling is most common in vessels of 150 micra in diameter or less. On the other hand a considerable number of capillaries are probably preserved in each specimen. In spite of the viscosity of the plastic material, vessels down to eight micra in size have been demonstrated by one of us (J. B. W.) in human specimens.

Some variation in the completeness of injection was noted when separate animals were compared. However, when the body weight of each animal in the control series was compared with the weights of the plastic casts of its hind limbs, no significant correlation could be found.

Variability in injection mass between paired limbs was minimized by cannulating the aorta above the iliac bifurcation.

A great deal of work remains to be done before the vascular response to fracture is clearly understood. In future reports the results of current studies as regards the effect of age on the vascular response, the relationships of the phenomena to bone union, and the vascular response to repeated refracture will be presented.

SUMMARY AND CONCLUSIONS

1. A measurable increase in the volume of the vascular bed of the hind limb of the rat occurs after a closed fracture of the tibia. The peak of the process occurs on the ninth day and appears to return to control levels with union of the fracture.

2. A technique permitting quantitative analysis of this process is described. Further application of the technique should contribute much to knowledge of the mechanisms of fracture healing.

NOTE: The authors are grateful to Dr. Maxwell Little, of the Department of Pharmacology and Physiology of the Bowman Gray School of Medicine, for his invaluable assistance in the organization of the experiment and the analytical interpretation of the data.

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Histological and Alkaline-Phosphatase Changes in Autogenous Transplants of Tibial Grafts in Dogs Treated with Cortisone*

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A better understanding of the effects of corticosteroids on the mineralization and repair of bone is necessary with the increasing use of these substances in patients with orthopaedic problems. The profound effects of cortisone on various metabolic activities were shown by Hench and associates and Layton. Some investigators [2,7,18,20] have believed that there is a delay in bone repair after treatment with compound E. Key, Odell, and Taylor, however, were not able to demonstrate this delay after cortisone. Dosage and animal resistance appear to be factors in this situation.

Duthie and Barker used rats in their studies. They administered one milligram of cortisone per fifty grams of body weight. Key, Odell, and Taylor failed to demonstrate any alterations in the repair of experimental fractures in rats, using cortisone in doses of about one milligram per fifty grams of body weight in one group, and about one milligram per 100 grams in another group. However, they did observe a persistence of hemorrhage at the fracture site in cortisone-treated rats. Blunt and associates, on the other hand, found rabbits to be very sensitive to approximately eight milligrams per kilogram of body weight of cortisone daily, whereas Curtiss did not find transplants in dogs to be particularly altered by one to two milligrams of cortisone per kilogram of body weight.

The treatment of rabbits with cortisone revealed an increased resorption of bone, whereas halting the treatment was followed by a rapid development of bone [19]. Storey [20] also noted that each new layer of bone was demarcated by a "reversal" line as a consequence of the repeated waves of resorption and deposition of bone after intermittent treatment with cortisone.

Duthie and Barker, studying fracture healing in rats with S^{35} autoradiograms, indicated that cortisone affects the formation of periosteal cartilaginous blastema and thus the process of endochondral ossification, with liberation of increased amounts of chondroitin sulphate which was calcified rather than ossified. Storey [20] also demonstrated that the effects of cortisone on cartilaginous growth in rabbits were that the long columns of metaphyseal cartilage were replaced by a layer of new bone, partly sealing the epiphyseal cartilage from the medullary canal.

Bone changes which have been described as a result of cortisone therapy lead one to suspect concomitant changes in alkaline-phosphatase activity. It was the object of this investigation to determine whether such changes occur.

MATERIALS AND METHODS

Eight adult male and female mongrel dogs, weighing from ten to twelve kilograms, were used in the experiments. The animals were divided into two groups: four control dogs and four cortisone-treated dogs. The cortisone-treated animals received an initial intramuscular injection of 100 milligrams, followed by fifty milligrams daily of compound E (the average dose per kilogram of body weight was two to four milligrams per day) for four weeks after operation.

* Read in part at the Combined Program of the Orthopaedic Research Society and The American Academy of Orthopaedic Surgeons, Chicago, Illinois, January 28, 1956.

A rectangular graft of cortical bone (two by three centimeters) was removed from the upper half of the shaft of each tibia and transplanted to the opposite leg. Two weeks after the first operation, a large rectangular area, which incorporated the previous healing transplant, was removed from the left tibia for tissue study. The right tibia was similarly operated on four weeks after the first operation. Sixteen specimens were available for the histological and histochemical studies. Roentgenograms were made of each leg prior to each operation.

Freshly removed surgical specimens were cut into smaller pieces to ensure proper penetration of the fixing agent. The samples were fixed immediately in 80 per cent alcohol for eight hours and decalcified by the Lorch method, a citrate-hydrochloric acid buffer of pH 4.5 being employed. The decalcified tissues were routinely dehydrated, embedded with paraffin for two hours at 54 degrees centi-

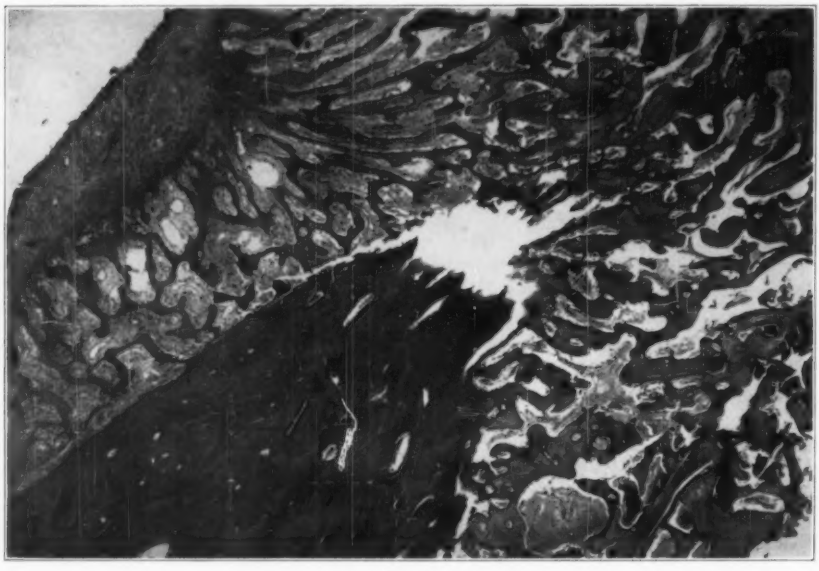

FIG. 1

A section of the tibia of a control dog in the two-week-postoperative group, revealing the active incorporation of the autogenous transplant. The periosteum is engaged in the abundant production of trabeculae. The graft reveals empty lacunae (hematoxylin and eosin, × 50).

grade, and cut at eight micra. Tissue sections were stained routinely with Harris hematoxylin and eosin for histological studies and by the revised cobalt method of Gomori [11,12,13] for alkaline-phosphatase activity. A sodium beta-glycerophosphate substrate of pH 9.5 was used in which the tissue sections were incubated at 37 degrees centigrade for one-quarter, one-half, one, two, four, eight, and sixteen hours, respectively. Enzyme activity was evaluated by employing a minimal staining technique [21]. Semiquantitation of alkaline-phosphatase activity was based on visual inspection of numerous sections prepared from each tissue sample available and on the assignment of arbitrary plus values. The difference between values in activity selected was large enough to prevent the possibility of confusion in the proper assignment of plus values.

RESULTS

Control Group

The two-week-postoperative group: Suitable consolidation of the grafts was observed at this stage grossly. Histological sections exhibited the usual findings

FIG. 2

A section similar to that of Fig. 1, revealing the activity and distribution of alkaline phosphatase. The osteogenic layer of the periosteum and the cells of the intertrabecular spaces, especially the osteoblasts, reveal a high enzyme activity, whereas the graft is devoid of alkaline-phosphatase activity at this level of detection (modified Gomori cobalt method, × 50).

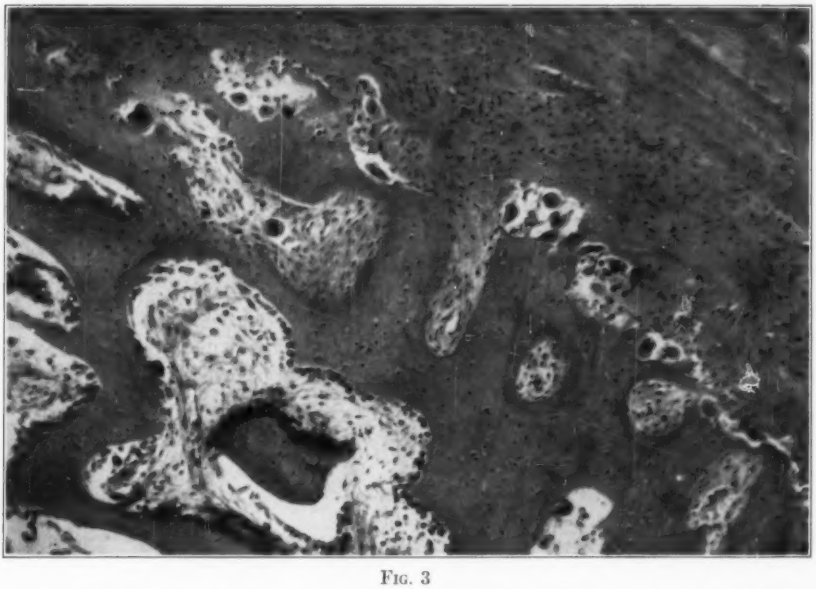

FIG. 3

A photomicrograph of the area of new-bone formation in a dog in the four-week-post-operative group, revealing the usual picture at this time of increased osteoclastic activity in the subperiosteal areas and the remodeling of trabeculae brought about by the invasion of capillaries and osteoblasts from the periosteum (hematoxylin and eosin, × 430).

of incorporation of a dead graft by the host. The abundant trabeculae that were formed are demonstrated in Figure 1. The osteogenic cells and matrix of the periosteal callus revealed a slightly positive reaction for alkaline phosphatase. This reaction was also found in the fibrous layer of the periosteum. The osteogenic layer of the periosteum, on the other hand, showed greater activity. As the distance from the graft junction to the lateral regions of the callus increased, the levels of alkaline phosphatase also increased.

The cellular components of the new cancellous bone forming about the ends of the graft showed some alkaline-phosphatase activity; the bone graft itself, however, was completely negative except for the blood vessels (Fig. 2).

The four-week-postoperative group: At this period there was the usual histological picture of abundant osteoclastic activity and remodeling (Fig. 3). The

FIG. 4

A section taken from a dog treated with cortisone in the four-week-postoperative group, revealing a reduced periosteal appositional bone formation and graft incorporation (hematoxylin and eosin, × 50).

periosteal callus exhibited an enzyme activity similar to that found in the two-week-postoperative group, but now the undifferentiated mesenchymal cells, which were present at two weeks, had been replaced by cancellous bone. At sites of active resorption and deposition, the osteoblasts were strongly positive, whereas the osteocytes revealed moderate phosphatase activity. The numerous osteoclasts which appeared at this stage were moderately positive for phosphatase activity.

Cortisone-Treated Animals

The two-week-postoperative group: Histological sections revealed a lack of graft incorporation or, at most, only partial incorporation at this stage; this was also observed grossly. Trabecular bone formation appeared to be minimal (Fig. 4). The numerous and characteristic osteoblasts which were found surrounding the trabeculae (Fig. 3) in the control group were encountered only occasionally in the cortisone-treated dogs. The sparse cellular population of these intertrabecular spaces appeared to be made up primarily of fibroblastic elements. Dense fibrous connective tissue appeared to have infiltrated the osteogenic layer of the periosteum. Only occasional small foci of new-bone formation were seen at the periphery of the graft.

Alkaline-phosphatase activity was greatly reduced throughout all the tissue sections in the cortisone-treated animal (Fig. 5). This was easily noted by gross

FIG. 5

A section similar to Fig. 4, studied for alkaline-phosphatase activity and distribution. Note the drastic reduction in enzyme activity throughout the photomicrograph and especially the periosteum, which is indicated by the arrow (modified Gomori cobalt method, × 50).

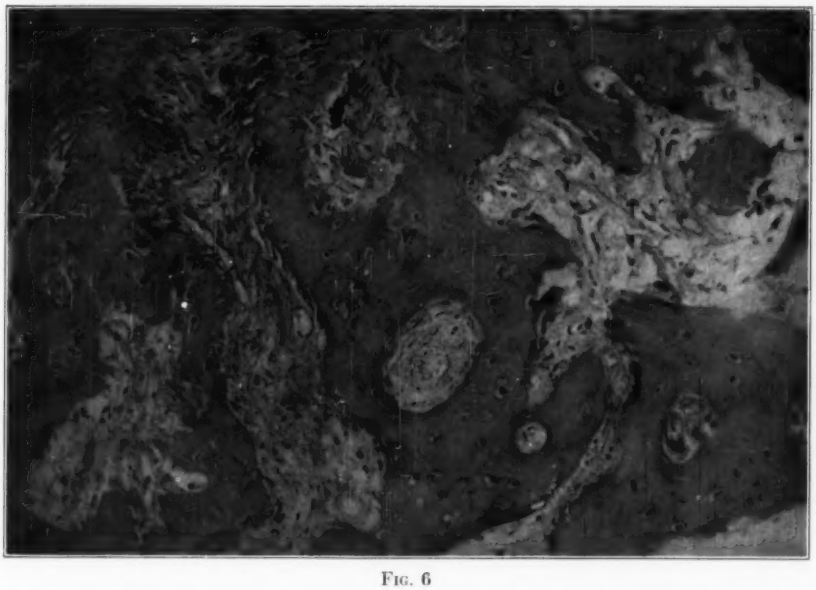

FIG. 6

An enlarged photomicrograph of a region taken from a dog treated with cortisone in the four-week postoperative group, showing poorly ossified trabeculae and a lack of sufficient osteoblasts and osteogenic cells making up the intertrabecular spaces. The morphology of the cells usually associated with normal trabecular formation and remodeling is missing (hematoxylin and eosin, × 430).

TABLE I

HISTOLOGICAL AND HISTOCHEMICAL OBSERVATIONS ON AUTOGENOUS TIBIAL GRAFTS IN CONTROL AND CORTISONE-TREATED (COMPOUND E) DOGS TWO AND FOUR WEEKS AFTER SURGERY

Observation*	Two Weeks after Operation		Four Weeks after Operation	
	Control Dogs	Treated Dogs	Control Dogs	Treated Dogs
Abundance of osteogenic cells	+++	+	+++	+
Abundance of connective tissue	+	+	+	+
Abundance of cartilage	±	±	±	±
General vascularity	++	++	++	++
Trabecular formation	++	±	+++	++
Abundance of osteoblasts lining intertrabecular spaces	+++	±	+++	±
Graft incorporation	+++	±	+++	++
Over-all alkaline-phosphatase activity	++	±	++	±
Periosteal alkaline-phosphatase activity	+++	±	+++	±

* +++ = high; ++ = moderate; + = low; ± = very low.

inspection of the slides, before examining them under the microscope.

The four-week-postoperative group: The over-all histological picture in the dogs in the four-week-postoperative group resembled to some extent sections from control animals at about two weeks after surgery. The tibial grafts which were now in the process of incorporation revealed increased trabecular formation. Active osteoblasts were rarely encountered about the surfaces of the trabeculae (Fig. 6).

Alkaline-phosphatase activity at this stage was only slightly elevated over that in the dogs in the two-week-postoperative group treated with cortisone.

A comparison of the findings in the groups at two weeks and four weeks after operation is given in Table I.

DISCUSSION

The healing of autogenous tibial grafts in dogs treated with cortisone lagged behind that in the control animals by about two weeks. This retardation is in agreement with the results obtained in other animals by several investigators [7,20]. The retardation of appositional periosteal growth was well demonstrated by the presence of "reversal" lines in rabbits treated intermittently with cortisone [20]. Duthie and Barker demonstrated in roentgenograms of the fractured tibiae of cortisone-treated rats that calcium salts of poor density were laid down. These authors believed that the calcium salts were deposited in the free chondroitin sulphate at the fracture site so that calcification rather than ossification was taking place. Although the roentgenograms indicated that fracture healing was taking place, actual consolidation of the fracture site by new-bone formation had not occurred. We have also observed this lack of graft incorporation in all cortisone-treated animals two weeks after surgery. It is interesting to note that newly formed trabeculae in the animals treated with cortisone in the four-week-postoperative group stained lightly with eosin, rather than with the deep red stain that occurs with properly ossified trabeculae. Even more striking was the absence of the large active osteoblasts, which are usually seen surrounding the newly formed trabeculae, and the sparse cellular complement of the intertrabecular spaces. The abundance of trabeculae normally seen two weeks after fracture was not encountered until four weeks after operation. Grossly, grafts from the cortisone-treated animals were not fixed to the surrounding bone two weeks after surgery. They could be removed easily from the defect. The lack of incorporation was clearly shown in the roentgenograms. True incorporation did not occur until four weeks after trauma.

The new bone forming about the graft of the cortisone-treated dogs exhibited a very low alkaline-phosphatase activity. This finding suggests that cortisone treatment with an adequate dosage during bone repair produces diminished alkaline-phosphatase activity. The important normal participation of this enzyme in bone formation is also reduced, and may be associated with the retardation of fracture healing. Normally, one expects an over-all increase in alkaline-phosphatase activity at the fracture site. The association of phosphatase activity with bone formation is well known [1,8,9,10]. It was shown by Bourne [3,4,5] that within twenty-four hours after trauma to the femora and skulls of guinea pigs, the periosteum about the injured area exhibited intense phosphatase activity. Tonna [22] further showed that injury to a rat's femur, even in old age, resulted in an elevation of alkaline-phosphatase activity. Trauma to the bone during cortisone therapy, however, was associated with a fall in phosphatase activity below the level of non-traumatized animals not receiving cortisone.

It appears from the results obtained in dogs, that heavy dosages of corticosteroids do have some early loosening effect on the transplants, although healing still did occur. We have found that cortisone used in doses of up to 1,000 milligrams per day for months in a patient with pemphigus produced profound and rapid demineralization in the spine and in the humeral and femoral heads. Such so-called cortisone-produced osteoporosis has been observed in other conditions also—notably, in rheumatoid arthritis after long-term treatment with corticosteroids and in Cushing's disease.

Since cortisone evidently reduced the alkaline-phosphatase activity at the sites of trauma in this series of dogs as well as in rats, it seems probable that so-called cortisone osteoporosis may be effected through this mechanism in the human being. No evidence exists as yet, however, to prove this. We have observed a number of fractures in patients receiving cortisone therapy in dosages comparable to those given to these dogs. All these fractures healed uneventfully.

SUMMARY

Cortisone treatment of dogs after transplants of autogenous tibial grafts revealed sharply reduced alkaline-phosphatase activity two and four weeks after surgery. Graft incorporation and suitable trabecular formation were not seen until four weeks after transplantation in corticosteroid-treated dogs; the trabeculae of the callus lacked the usual osteoblastic lining. Histologically and grossly, the graft site in cortisone-treated dogs exhibited a retardation in bone healing of two weeks when compared with that in control dogs.

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Presidential Address*

BY C. LESLIE MITCHELL, M.D., DETROIT, MICHIGAN

Presidential addresses to the Association in the past have been on a wide variety of topics but perhaps they can be classified as being, in principle, either historical, philosophical, scientific, or political. All have been sincere and have conveyed a message, and many have been critical of the course the Association was taking or of its shortcomings and have attempted to chart a better course.

My address has no title: the subject matter is not scientific, nor is it political; however, it would be difficult to deliver an address of this type without delving somewhat into our history and without waxing a bit philosophical. I suppose if I did have a title it would be, "The Value of the Team Approach in the Practice of Orthopaedic Surgery".

Historically, I will refer back a quarter of a century ago when it was my privilege to attend, as a guest, my first meeting of the Association. It was my good fortune to attend the meeting at which Dr. Arthur Steindler was presiding and to have the opportunity of hearing his Presidential Address. His address also did not have a title, but it was thought-provoking and stimulating and dealt with the problems of biomechanics and pathomechanics of locomotion and emphasized the need for study in the field of energy consumption in pathological motion. To quote:

"I visualize biomechanics as a powerful and indispensable ally of the orthopaedic clinician. Many of my colleagues, I trust, will be attracted by the intriguing problems in this field, and I hope to see the day when systematic and coherent research in the domain of biophysics of locomotion will open the way for greater proficiency and will mark a definite advance in orthopaedic surgery."

Dr. Steindler in his address thus touched on the team approach to an important problem of interest to all of us. In the past twenty-five years, there has been an awakening in the field of orthopaedic surgery, as in other medical specialties, to the fact that, because of the growth of medical knowledge, it is no longer possible or practical for the individual physician to do justice to his patient or to medical advancement in general without close cooperation with those in allied fields.

We must not disparage or discourage the individual physician working independently who by pursuing well charted courses or by serendipity arrives at an important discovery or observation. Certainly this has been, by and large, the history of great medical discoveries in the past. Men such as Pasteur, Jenner, Fleming, Hunter, Banting, and others were great individualists and contributed so much to our present knowledge of disease and therapy. Undoubtedly, we will continue to have these giants in medicine and nothing should be done that might discourage the fostering and developing of such individuals.

Medicine, however, has become so much more complex in the last few decades that it is almost impossible for a single individual to work independently on a problem without a thorough knowledge of its background and without the cooperation of others in allied fields. In many instances the specific problem has been obvious but the individual approach or solution to it has been difficult, if not impossible. A team, therefore, working toward a common goal has been shown often to accomplish dramatic solutions of many of our problems. This team may consist of several individuals or groups with similar training and

* Presented at the Seventy-second Annual Meeting of The American Orthopaedic Association, Lake Placid, New York, June 17, 1959.

background but with different ideas of approach to the problem at hand or it may more frequently and effectively consist of individuals with different training backgrounds in allied fields.

Our specialty, more than any other, has been closely allied with the field of engineering and yet how rarely in the past have we sought the help and cooperation of competent engineers in helping to arrive at a solution to so many of our problems. The trend is fortunately now changing and many research projects are being conducted jointly with engineers, physicists, biochemists, and others, who because of their background and knowledge, have an approach to our problems that is of inestimable help to us. Thus the metallurgist has assisted us in the development of materials inert in the human body; thus the mechanical engineer has taught us principles of stress, strain, and torsion and the effects of corrosion and fatigue; thus the physicist has taught us methods of studying bone, cartilage, and muscle structure; thus the biochemist has taught us the fundamentals of bone, muscle, cartilage and collagen physiology, and principles of protein, carbohydrate, and hormone chemistry, and the details of mineral metabolism.

From all of these and many other disciplines we have profited a great deal and we are learning to take advantage of their body of knowledge and apply it to our special problems and work closely in conjunction with them.

During World War II and immediately following it, it became increasingly apparent that although tremendous strides had been made in the treatment of the wounded and in decreasing morbidity and mortality, a great deal was left to be desired in the total rehabilitation of many of the patients. This was particularly true in the case of the amputee, and it soon became apparent to many, especially the amputee himself, that little, if any, progress in this field had been made for many years as compared with the remarkable advances in so many other medical and surgical conditions.

Thus was stimulated an interest in investigating and in improving prostheses, and acting on a suggestion of the Secretary of War and under the auspices of the Surgeon General, the National Academy of Sciences, and the Veterans Administration, a committee was appointed to investigate the problem, with funds being appropriated by Congress.

The original committee was composed of outstanding orthopaedic surgeons and engineers and it soon became apparent to them that before any worthwhile contributions could be made, it was necessary to have more knowledge about the fundamentals of locomotion, gait, and biomechanics and energy consumption than was available.

The next step was to select key individuals with the interest, background, knowledge, and devotion in the field of biomechanics to carry out investigative work. By a happy circumstance, a group at the University of California was already interested in this problem and under the direction of Dr. Verne Inman of the Department of Orthopaedic Surgery, and Professor Howard Eberhart of the Department of Engineering, a concentrated program of biophysics, biomechanics, and gait study was undertaken. As a result of this study, which is still continuing, and organized research work at other university centers, invaluable information has been obtained in the care of the amputee, including better knowledge of normal gait, better construction and fitting of the prosthesis, and improved teaching in its use. These studies naturally led to the investigation of energy consumption which has been of prime interest to this group of investigators and which will be applicable to many other problems of locomotion.

Thus we have a directed team approach to an important problem in our field which has borne fruit, and the information gained so far has been of inestimable value to the patient and to the orthopaedic surgeon. Similar studies have been carried out at other centers, principally the University of California at Los Angeles, on the problem of upper-extremity amputees, and the information

gained by this team approach to the problem by engineers, orthopaedic surgeons, and other disciplines, has been made available to all those vitally interested in the care of the amputee.

In Michigan, at Grand Rapids, some few years ago, under the auspices of the Michigan Crippled Children Commission, a concentrated program for the study and treatment of the juvenile amputee was instituted under the direction of Dr. Charles Frantz and Dr. George Aitken. This joint effort, which had the cooperation of the Children's Bureau and the Prosthetics Research Board of the National Academy of Sciences, has resulted in a tremendous advance in our knowledge of a problem about which very little was known heretofore. This also represents a team approach toward a problem with the use of many disciplines working toward a common goal.

This method of approach to a common problem seems to me to be, in part, at least, an answer to Dr. Steindler's plea that systematic and coherent research in the domain of biophysics and biomechanics of locomotion be undertaken. The avenues opening up from these fundamental studies are innumerable, and in our imagination we can see it applicable to so many allied problems, such as the poliomyelitic, paralytic, paraplegic, and many congenital and developmental abnormalities. Improvements in bracing including equipment, motivation, external power, and training, have all become the natural outcome of this organized investigation.

Team approach to orthopaedic problems, however, is by no means limited to disciplines outside of the field of medicine itself. We are fortunate in that our specialty, which anatomically embraces such a large portion of the human body, brings us into close contact with so many other fields of medicine including endocrinology, metabolism, rheumatology, vascular diseases, and neurology—to mention only a few. The role of the orthopaedist has never been narrow, inasmuch as it encompasses lesions of the bones, joints, muscles, tendons, nerves, and blood vessels of the extremities and trunk. The well trained orthopaedist, therefore, has to be familiar with a multitude of conditions affecting these parts. These conditions include lesions that are in nature congenital, developmental, traumatic, inflammatory, structural, metabolic, endocrinological, vascular, neurological, neoplastic, functional, and dermatological. It is necessary that the orthopaedist be aware of all of these possibilities in the differential diagnosis; but, because of the complexities of medicine today, it is not possible for the individual physician or surgeon to do justice to the patient in the treatment of all of these disorders. Therefore, in the best interest of his patient it is often advisable for the orthopaedist to work in close conjunction with other medical disciplines, keeping always in mind the total care of the patient and not discharging the patient from his responsibility and care unless it is decided that it is in the patient's best interests that he be transferred to the care of one of the other medical specialties.

As Dr. Steindler and many others have stated, we must be physicians first, surgeons next, and finally orthopaedists. Perhaps we should be designated orthopaedic physicians rather than orthopaedic surgeons inasmuch as such a high percentage of patients consulting us have non-surgical problems. We obviously have as great a responsibility in the care of the non-operative patient as of those with operative conditions; and the rewards are just as great, and in many instances greater, if we assume leadership in the total care of the patient. I am afraid that there are too many of our younger orthopaedic surgeons who lose interest in a patient if no surgical possibility suggests itself. This attitude, of course, is to be deplored and reflects adversely on the prestige of our specialty. As the late, beloved Dr. Bost stated, "The duty of orthopaedic surgeons requires our constant and alert attention to the progress of our intermedical relations and to the sociomedical and medical-economic aspects of our specialty". As Dr. Caldwell stated, "probably more than half the crippling conditions which affect man-

kind are amenable to non-operative orthopaedic measures alone and these patients deserve as much thoughtful care from us as do those who, in addition, require some operative procedure".

If we in orthopaedic surgery are to progress and to continue to hold our present position of prestige in the medical profession, it is not enough that we treat the obvious condition that the patient presents himself with but that we, in many instances, assume "the total care of the patient". Unless we do so, we are in danger of being considered mechanics or technicians at the beck and call of the pediatrician, the internist, the general practitioner, and in many instances, lay organizations. In other words, we should assume leadership in the care of our patient and consider the patient as a whole and work as a team with other disciplines toward the total care of the patient which, of course, includes socio-economic as well as physical rehabilitation.

Thus, in many centers it has been found advantageous to hold clinics at regular intervals for such conditions as poliomyelitis, cerebral palsy, amputation, and paraplegia. The combined approach to clinical problems in common to multiple medical and ancillary specialties cannot fail to promote improvement in the over-all knowledge of the patient's problems, and will thus lead to better therapy, and less confusion and misunderstanding will be conveyed to the patient and the patient's family. Leadership in most instances should be assumed by the orthopaedist. Thus, in the Amputee Clinic the patient is seen with the orthopaedist, the physiatrist, the prosthetist, and the therapist. The patient is evaluated by the group; prescriptions are written for the prosthesis; the prosthesis is evaluated; training is instituted; the gait and function are analyzed; and, when training is completed, vocational rehabilitation is undertaken when necessary, with the help of social service or other agencies. This is carried out in conjunction with the internist or surgeon referring the patient and constitutes an example of the team approach to the problem and the total care of the patient.

The character and type of orthopaedic practice has changed a great deal in the past twenty-five years. Conditions such as tuberculosis, osteomyelitis, poliomyelitis, and deficiency diseases are gradually passing from the picture. Today more than ever before we are dealing with more problems associated with trauma and geriatrics. Because of increased mechanization and speed of travel in the past twenty-five years, trauma has increased; because of the increase in life expectancy and span of life, there has been a marked rise in the population of elderly people with the various degenerative diseases incidental thereto. Thus we have gradually inherited more and more the care of the injured patient and many of the infirmities of the aged, and it is our obligation to accept this responsibility and to assume leadership where, frequently, it is indicated.

The increased demand for orthopaedic surgeons in America in the past twenty years and especially in smaller communities has been due largely to the fact that trauma to the spine and extremities has been accepted by the public and the profession at large as being in the domain of the orthopaedic surgeon. This is due to many factors including undergraduate teaching in medical schools where instruction in fractures and trauma to the extremity is carried out in a high percentage of institutions by the orthopaedists. This is due also to the influence of World War II and the impact it had on such a high percentage of our population that lesions of this type were so well handled by orthopaedists and to the fact that today most hospitals throughout our country call on the orthopaedist for the care of the severely injured patient.

Thus, we have by an evolutionary process inherited the role of traumatologist which we have assumed in addition to our many other responsibilities. We are, therefore, presented with another tremendous challenge, that is, of accepting leadership in this field, of improving our training program in traumatology, and of working in close harmony with our medical and surgical confreres in

closely allied fields. Traumatic lesions are by no means limited to orthopaedic conditions, but no one individual can possibly be an expert in all fields of traumatology. Therefore, again the importance of a team approach to a problem is obviously essential for the ultimate welfare of the patient, and all specialties necessary should be involved in the care of a severely injured patient. Often the orthopaedist is asked to take charge of the over-all care of the patient, and he should be ready at all times to assume leadership and to call on other disciplines for help in the care. In these circumstances the orthopaedist in accepting the responsibility for the total care of the patient must be familiar with signs and evidence of trauma to areas not immediately associated with lesions in his domain. Only in this manner can we avoid the stigma of being known as isolated specialists and can we help to dispel any criticism from the patient, the public, or the profession, that might be directed at us that we are not willing or able, when necessary, to assume responsibility for the total care of the injured patient.

It is mandatory, therefore, that in our undergraduate and graduate teaching programs, sufficient emphasis be placed and adequate training be supplied, in all phases of trauma to the trunk and extremities. The trainee must be thoroughly provided with a knowledge of fundamentals in this field including bone physiology, biomechanics, shock, fluid balance, principles of reduction and immobilization, care of associated soft-tissue injuries, bone-grafting, physical therapy, and rehabilitation.

Co-existent, however, with the gradual increase in the care of fractures by orthopaedic surgeons, is the ever-present danger that the term *orthopaedics* be considered by the laity and the medical profession at large as synonymous with the treatment of fractures. This danger can be circumvented if we constantly demonstrate our willingness and ability to work in close cooperation with our medical confreres in problems of mutual interest. We must impress the laity as well as the medical profession with our concern in the comprehensive non-surgical as well as the surgical treatment of chronic disease which leads to deformity and disability of the extremities. In addition, we must assume a role which has been neglected in the past by a large percentage of orthopaedic surgeons, namely, that of the total care of the patient with supervision through the complicated processes of rehabilitation to maximum physical, mental, and social recovery. If we do not assume this role, or if we do not evince sufficient interest in non-surgical orthopaedic problems, we are in great danger of having these patients and their problems absorbed by other medical disciplines.

Great strides have been made in the past few years in the progress of orthopaedic surgery because of stimulation by leaders in our field and by improved instruction in the research and clinical aspects of our specialty. Much credit is due to the American Board of Orthopaedic Surgery for stimulating improvement in our training programs, for their surveillance of existing training programs and accreditation of proposed facilities. The Board, composed as it is of representative members from The American Orthopaedic Association, The American Academy of Orthopaedic Surgeons, and the Orthopaedic Section of the American Medical Association, is democratic in that it encourages and embraces ideas from all orthopaedic surgeons for the betterment of orthopaedic surgery in the country.

The recent meeting of the Board in Chicago relative to residency training—which was so well organized and attended by so many leaders in the field of orthopaedic surgery—was testimony to the fact that all in the teaching fields are vitally interested in continuing to improve the training programs. This was a healthy and stimulating program and indicated that, although very great strides have been made in the past, we are not satisfied with our accomplishments and are seeking ways and means to further improve our programs. The American Board of Orthopaedic Surgery was originally designed as an examining board, with certain criteria of training and training programs being set

up as standards for Board eligibility. Accreditation of training programs became the joint responsibility of the Board and the Committee on Graduate Education of the American Medical Association. Periodic inspection of existing training programs has been carried out jointly by these two organizations, as well as inspection and decision regarding proposed resident training programs. Information gained by the Board over the years as a result of Board examinations, Part I and Part II, has been invaluable in assessing the various training centers and in improving the standards of the various programs throughout the country. However, the question has arisen as to whether we have now reached the point where the Board should be strictly an examining body and that accreditation and investigation of training programs should be performed by a separate committee composed of members selected by The American Orthopaedic Association, The American Academy of Orthopaedic Surgery, and the American Medical Association. This last committee could have information made available to them by feedback from the results of the Board examination that are relative to the various training programs. Although existing training programs are not and should not be standardized, I believe that the Board with its Residency Review Committee with representatives from The American Orthopaedic Association, The American Academy of Orthopaedic Surgery, and the Orthopaedic Section of the American Medical Association, is the logical body to carry out this function. This has been conducted successfully by other similar specialties and I feel that we should give it a further trial before considering changing it.

Emphasis in the past on the training programs was on therapeutics with particular stress on surgical and mechanical approaches to problems in our field. In our present training program much more emphasis is being placed on the teaching of fundamentals, the stimulation of research and training in allied medical and ancillary disciplines. Insufficient emphasis, however, has been placed on the total care of the patient including the sociomedical and medical-economic aspects of our specialty. Whether or not we are in accord, we must face the fact that there is an increase in paternalism directed toward the health and welfare of the crippled and disabled. Government agencies, as was stressed in Dr. Eaton's Presidential Address last year, are gradually assuming control of the care and rehabilitation of the disabled patient. Private organizations for many years have been interested in the welfare of the crippled child and disabled adult. In many instances care is available for those who would otherwise be deprived of it.

In our training program, therefore, we should include information and instruction in the economic facilities available for the rehabilitation of our patients who would otherwise be deprived of total medical care. Through social-service facilities the trainee can be taught what economic resources can be approached for the total care of deserving patients—whether this be for therapy, braces, prostheses, or vocational guidance.

In order that the resident obtain the maximum benefit from his training program, it is desirable, if not essential, that he have the opportunity of observing and helping in the office care of private patients, under the supervision and direction of his instructors. Thus, he will not only be able to observe and to develop the art of medicine, but he will have an opportunity of seeing and evaluating many patients with problems not requiring hospitalization or those who require a multidisciplinary approach. Having observed and helped in the handling of patients in this manner, he will find that the transition from his training program to the private practice of orthopaedic surgery is effortless and that minimum adjustments will have to be made.

With the advent of prepaid insurance plans, and various third-party interests, there are relatively few indigent or charity cases available for undergraduate or graduate training. Training of residents on a private-patient basis has been conducted successfully in many institutions for a considerable period of

time, and I feel that we need have no fear of a dearth of teaching material or facilities because of the socioeconomic change. We have been able to carry on successful graduate training in all specialties at our own institution for over forty years on a private-patient basis—this experience has been duplicated in the past at many similar institutions.

Great changes have taken place in the past twenty-five years in the practice of orthopaedic surgery and in the training of our residents. Unquestionably great advances have been made in the training programs, the clinical approach to orthopaedic problems, and in conducting and stimulating basic and clinical research in our specialty. However, although great advances in the progress of orthopaedic surgery have been made in the past and will continue to be made in the future by individuals of stature in our profession working more or less independently, we must recognize the importance of the team approach to problems of mutual interest in research, clinical investigation, and therapy.

We must accept and assume our rightful role as leaders in the over-all care of patients with problems primarily of an orthopaedic nature but be prepared to cooperate to the fullest with other medical specialties involved.

We should forever strive to take advantage of knowledge and assistance from other medical or scientific sources for conducting of research projects and for improvement in the clinical treatment of our patients. In our training programs we should provide an opportunity for our residents to grasp a firm knowledge of the sociomedical and medical-economic aspects of our specialty and instill in them the idea of the team approach and the total care of the patient.

In conclusion, may I take this opportunity of expressing to the members of the Association my deep appreciation of the great honor they have bestowed on me by naming me their President. I have an unbounded faith in the future of our Association and it is my fervent hope that in some small measure I have helped to direct the proper charting of its course.

News Notes

THE JOURNAL OF BONE AND JOINT SURGERY, BRITISH VOLUME 41-B, No. 3

Publication of the August issue of the British Volume of The Journal has been delayed by the printers' strike in Great Britain. The dispute is now settled, but publication date will be delayed six to seven weeks. The London Office wishes to apologize for this unfortunate yet unavoidable delay.

At the recent meeting of **The American Orthopaedic Association,** Dr. John Royal Moore, Philadelphia, Pennsylvania, was inducted into office as President. The following officers were elected:

President-Elect: Edwin F. Cave, M.D., Boston, Massachusetts;
Vice-President: Alan DeForest Smith, M.D., New York, N. Y.;
Secretary: Lee Ramsay Straub, M.D., New York, N. Y.;
Treasurer: Mather Cleveland, M.D., New London, New Hampshire.

The following were elected to membership:

Randolph Anderson, M.D., Charleston, West Virginia;
William F. Donaldson, M.D., Pittsburgh, Pennsylvania;
William R. Duncan, M.D., Seattle, Washington;
James T. Green, Jr., M.D., Columbia, South Carolina;
John W. Hillman, M.D., Nashville, Tennessee;
J. Gordon Petrie, M.D., Toronto, Ontario, Canada;
Ira H. Rapp, Charlotte, North Carolina;
Howard B. Shorbe, M.D., Oklahoma City, Oklahoma;
Donald E. Starr, M.D., Vancouver, British Columbia, Canada;
Arthur Thibodeau, M.D., Boston, Massachusetts;
Frederick Webster, M.D., Lincoln, Nebraska;
Jack K. Wickstrom, M.D., New Orleans, Louisiana;
Leon L. Wiltse, M.D., Long Beach, California.

The Twenty-seventh Annual Meeting of **The American Academy of Orthopaedic Surgeons** will be held at the Palmer House, Chicago, Illinois, January 23 to 28, 1960.

The Annual Meeting of the **Western Orthopedic Association** will be held at the Brown Palace Hotel, Denver, Colorado, October 18 through 22, 1959.

A one-year **fellowship in orthopaedic pathology** will be available at the Ochsner Foundation Hospital beginning on July 1, 1960. For information, write to Mary S. Sherman, M.D., Ochsner Clinic, 3503 Prytania Street, New Orleans 15, Louisiana.

At the last meeting of the Board of Trustees of **The Orthopaedic Research and Education Foundation,** it was decided to increase the number of Board Members from six to eight. Dr. Harold B. Boyd, Memphis, Tennessee, has been named to represent The American Academy of Orthopaedic Surgeons, and Dr. George W. N. Eggers, Galveston, Texas, has been named to represent The American Orthopaedic Association. At this meeting the following officers were elected:

President: Joseph S. Barr, M.D., Boston, Massachusetts;
Vice-President: James A. Dickson, M.D., Cleveland, Ohio;
Secretary-Treasurer: Harold A. Sofield, M.D., Chicago, Illinois.

Dr. A. R. Shands, Jr., Wilmington, Delaware, was named Chairman of the Development Committee.

Solicitation for funds will start in the early fall and will continue through until the Academy meeting in January. Support from every orthopaedic surgeon is earnestly needed in this first intensive campaign to raise the much needed funds to help in the continued development of orthopaedic surgery.

The Sixth Annual Meeting of the **Mid-Central States Orthopaedic Society** was held in Omaha, Nebraska, April 9 through 11, 1959, under the Presidency of Werner P. Jensen, M.D. The following officers were elected to serve for the ensuing year:

President: H. O. Anderson, M.D., Wichita, Kansas;
Vice-President: Daniel Yancey, M.D., Springfield, Missouri;
Secretary-Treasurer: H. O. Marsh, M.D., Wichita, Kansas.

The Seventh Annual Meeting of the Society will be held in Wichita, Kansas, at the Lassen Hotel, April 7 through 9, 1960.

The Journal takes pleasure in announcing the formation of the **Sociedad Nicaraguense de Ortopedia y Traumatologia.** The officers of the Society are: Dr. Horatio Castellón, *President;* Dr. Luis Ode, *Vice-President;* Dr. Juan E. Zelaya, *Secretary;* Dr. Leandro Pasos, *Treasurer.* A warm welcome from the Society awaits any visiting orthopaedist from north or south. The Secretary's address is: Calle del Tiunfo No. 305, Managua, D.N., Nicaragua.

FREDERIC CARROLL BOST, M. D.
1900-1959

To write an obituary is a sad privilege at best, for it marks the passing of a fellow traveler on life's paths. The attempt, in any obituary, to epitomize the soul of an absent friend makes heavy demand on the chronicler, who can hope only to see clearly a few facets of the true total personality. With this in mind, several other friends have been drawn into this effort of love and respect, with the hope of supplementing my viewpoint. These men have all emphasized the same attributes, enlarging on certain points, but with a common theme: Toby Bost will never be forgotten by anyone whose path crossed his in life, for no matter what the occasion, he left a positive mark on their memory. Perhaps Toby Bost's most outstanding trait was love—love for his wonderful family and a keen interest in the problems of each and every one. He was devoted to his wife Jean, and to his five children—Gail, Alice, Frederic, Cindy, and Bettina. He was equally devoted to his brother Abe, his parents, and other members of his immediate family. His St. Francis Woods home, and later his home in Belvedere were always open to his friends in California and throughout the world. During World War II he entertained myriad soldiers who passed through San Francisco, either on their way to, or from the States, and who had been uprooted from their homes and loved ones.

Probably no orthopaedist in the world had more genuine friends than did Toby Bost. His was a rare ability to make you know that he liked you, and that he cared about your problem. In return for this warmth of interested insight, he was one of the most beloved in our ranks. He was a keen observer of people and what they stood for, not just what they said. He had no patience with dishonesty of deed or purpose. He stood at all costs for the right as he saw it and with a determination that could not be matched in unquestioned integrity, clarity of insight, and simple directness. This made him a master of eloquent persuasiveness. "The heart of the matter" was clear to him, and since this was so, it became

simple to interpret the situation to others, and to be forcibly direct in presenting the subject.

In professional matters of judgment regarding patient care few were his equal, and he was excelled by none. He had utter disdain for the cutting doctor, and although his unusual technical skill made him a most adept surgeon, as well as a highly proficient teacher—since he knew this facet so thoroughly—it is certain that basically he would much prefer to be remembered as the well rounded physician he was in every instance.

Toby Bost, Clinical Professor of Orthopaedic Surgery at the University of California School of Medicine, had much to do with the Department of Orthopaedic Surgery there, which was developed and nurtured by LeRoy Abbott. Toby's students will long recall his insistence on perfection, and his ruthlessness with those who fell short of his ideals. He was a keen observer of the problems of the patient, and although he was not of the era of the team approach, he needed no team, for he took time personally to do the many services for his patients that are now so often delegated to others. His was an acute penetration, and an uncanny knowledge, not only of the purely technical aspects of orthopaedics, but also of the social aspects that determined the environmental climate for each patient.

The many technical papers which he gave to the orthopaedic world were always given with a zealous determination to present the problem under consideration with honesty and clarity and in its entirety. Those works presented in final form represented only a small portion of his voluminous clinical concerns. His interest in clinical research was eager, and his ability to recognize value and sound reasoning in experimental material presented was profound. This led to his activity with the Research Committee of the Shriners' Hospital for Crippled Children, and to related activities at that hospital in San Francisco, where he was Chief Surgeon, having been appointed to this post in 1944.

His was a dedicated and jealous service to orthopaedics in its many and varied phases—clinical, experimental, political, and administrative. Certainly, we shall always feel a reverence for what he stood for, and for the devotion he displayed toward his work.

I knew him in his heartaches and in his successes; in his driving, unrelenting energy for work and play; and in his deep friendship and understanding. Never can another fill this place in our hearts.

And now to the less arduous portion in the task of the chronicler. Frederic Carroll (Toby) Bost was born in Nevada City, California, December 2, 1900. He attended the University of California, receiving his M.D. in 1926. This was followed by internship at the University of California Hospital in 1927, and one year's service at that hospital as Resident in general surgery in 1928. The following three years were spent as a Resident in Orthopaedic Surgery in Boston, with assignments at Massachusetts General Hospital and the Children's Hospital. He then returned to San Francisco, where he became associated with LeRoy Abbott, in the Department of Orthopaedic Surgery at the University of California School of Medicine. In 1944 he was appointed Chief Surgeon at the Shriners' Hospital for Crippled Children at San Francisco, the hospital where he had served as a student extern twenty-five years prior to his appointment as Chief, under Dr. Walter Baldwin. In 1946 he was a guiding figure in establishing the May T. Morrison Center for Rehabilitation, on whose Board he served with true devotion thence onward. As Chief of Surgery at Children's Hospital of San Francisco, he was responsible for the modernization of the physical plant of that hospital, and for the direction of important changes in the attitude toward the practice of medicine. In those societies whose chief function is the dissemination of the knowledge of orthopaedics, he served as President of The American Academy of Orthopaedic Surgery, as Chairman of the Section of Orthopaedics of the American Medical Association, and as a member of the Section of Orthopaedics of the American Medical Association, and as a member of countless committees working tirelessly for the betterment of orthopaedics. He was also a member of The American Orthopaedic Association, the Western Orthopaedic Association, The International Society of Orthopaedic Surgery and Traumatology (S.I.C.O.T.), Pacific Coast Surgical Society, The Wilson Interurban Orthopaedic Club, of which he was co-founder, American College of Surgeons, The San Francisco Medical Society, The California Medical Society, and the American Medical Association. He was a proud member of the Bohemian Club, Cercle de l'Union, and California Lodge No. 1, F. & A. M.

He died on February 2, 1959, at 3 a.m. He is survived by his wife Jean; his children, Mrs. Gail Rey, Alice, Frederic, Cindy, and Bettina Bost; his twin brother, Doctor Crawford Bost; and an aunt, Mrs. Carol Crawford of San Francisco.

As we review the life and works of our friend Toby Bost, we may each find love and guidance from this indomitable spirit portrayed and epitomized so well in his self-effacement, and his determination not for one minute to give in to physical infirmities; his unswerving desire to live each day for what it might bring—and to its very fullest. For him the beloved nickname of his close friends will always be preciously remembered—indeed, he died as he had lived—"The Little Giant".

E. R. S.

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Book Reviews

POLIOMYELITIS. Papers and Discussions Presented at the Fourth International Poliomyelitis Conference. Philadelphia, J. B. Lippincott Company, 1958. $7.50.

This volume is a compilation of the papers and discussions given at the Fourth International Poliomyelitis Conference held in Geneva, Switzerland, during July 1957. Its format in general follows that of previous editions in this series and is divided into sections which relate to various aspects of the disease. In general, this volume should be on the required reading list for all of those who deal with poliomyelitis, particularly during the acute and early convalescent phases; but from the standpoint of the orthopaedic surgeons, the latter portion of the book is of greater importance.

According to the reports of the official delegates, there is the same problem with the disease over the world as has existed in the past. The section on vaccination brings up to date the basic principles underlying the immunization procedure and also the knowledge regarding the oral vaccine which was known at that time. Indeed, the early section of the book is concerned with virus diseases of one sort or another including the ECHO and Coxsackie viruses. As is so often true, many of the problems presented in this section of the volume have been overcome in the year succeeding the publication of this particular material. However, the material is presented in such a way that even the novice in these diseases is able to obtain fairly good background in regard to this whole problem. The newer methods of diagnosis of the disease and the tests which are being performed are well covered in the section on diagnosis.

The experience and principles of therapy which have been obtained in the respiratory and rehabilitation centers of this country are well presented. The problems of basic respiratory mechanisms and the effect of paralysis are covered as are those of swallowing difficulties and the technique of glossopharyngeal breathing. The use of the various types of devices for artificial respiration, such as the tank respirator, positive pressure respirator, rocking bed and portable respirator with their indications and contra-indications, are described. In such severe diseases as represented by these patients, there are many disturbances in other parts of the body; these are summarized and the problems of management are outlined in the sections on the care of the patients severely stricken by poliomyelitis. Much of the material involving pulmonary complications, gastro-intestinal complications and genital urinary complications are applicable not only to the respiratory poliomyelitic patient but to patients with other severe debilitating diseases. Certainly the individual who has to care for these patients without too much previous experience can well benefit from these particular sections.

The basic principles concerned in the treatment of the patient with poliomyelitis are outlined in a simple and straightforward fashion. There is a considerable section on the problems of paralytic scoliosis. A classification of paralytic scoliosis is presented with which some may agree and others disagree but it appears to be a reasonable attempt to outline the problem of paralytic scoliosis and does furnish a working outline from which treatment can be derived. In general, the surgical treatment of paralytic scoliosis seems to follow accepted methods although in the discussion there is a mention of the use of metallic devices which exert a spring force to correct the scoliosis. Much of this work is still in the experimental and evaluation stage. There is a very useful section concerning the problems of severe paralysis in the upper extremities. In this, an outline of various surgical techniques and functional devices which can be used are well presented.

In general, there is considerable material in the second half of this volume which would be of considerable interest to the orthopaedic surgeon, particularly those concerned with the management and problems of the patient with severe paralysis. The techniques which are presented will doubtless find application in other conditions besides that of poliomyelitis.

Arthur L. Trott, M.D.

KLINICKA PROBLEMATIKA NIEKTORÝCH KOSTNÝCH NOVOTVAROV (Clinical Problems of Certain Bone Neoplasms). Published by the Sloven Academy of Sciences, The Section of Biology and Medical Sciences, under the Editorship of Professor M. J. Červeňansky. Bratislava, 1957.

The book, in the Slovakian language, represents a collective effort of a number of authors. The text—an important collection of valuable material—is divided into three parts: The first part comprises some general problems of histology, pathology, physiology, oncology, and treatment of tumors. The second part describes tumors characteristic of certain anatomical regions, and the third part reports case histories of special interest.

The text integrates gross and microscopic anatomy, physiology, various types of treatment, including surgery, and results obtained in a short and long follow-up study.

There are 213 illustrations in the text; 116 more illustrations, under the name of photographic documentation, are included in an addendum.

It is regrettable that this material is published in a language not accessible to larger groups of readers. A second edition of this volume by the Sloven Academy of Sciences could be of considerable help if each chapter were followed by a short résumé in English, French, or German.

Emanuel B. Kaplan, M.D.

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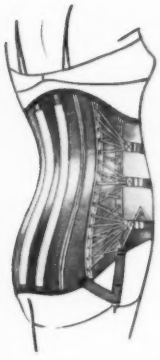

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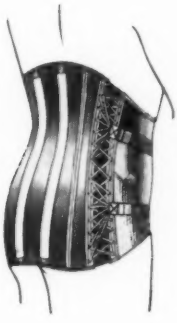

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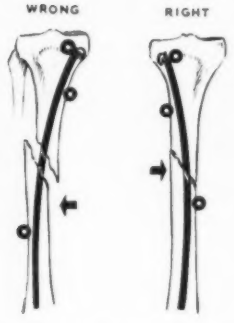
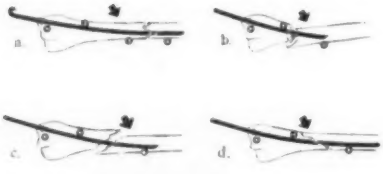


Direction of pin insertion determines compression or distraction.



V (victory) = Good Compression

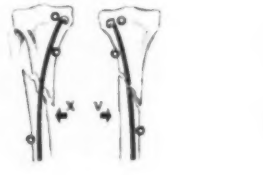
X (no good) = Distraction

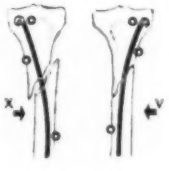
RADIUS

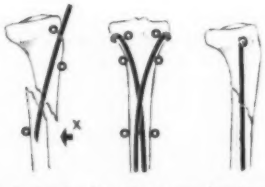
Length of fragment and direction of fracture line determine angle of pin insertion.

RULE

Axis of pin should be same direction as axis of fracture line, intersecting to make a "V" instead of an "X".







TIBIA

In fracture (lower left), double pins required because "V" cannot be accomplished.

* Rush, Leslie V.; Fracture Problems, Mississippi Doctor, Vol. 36, No. 10, March 1959.

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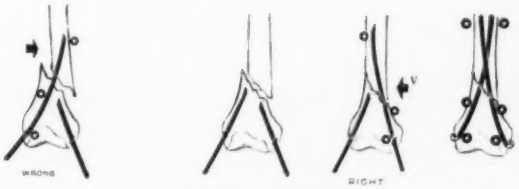
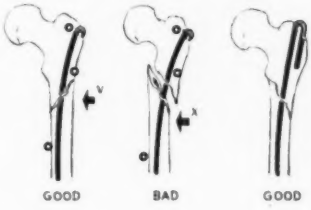


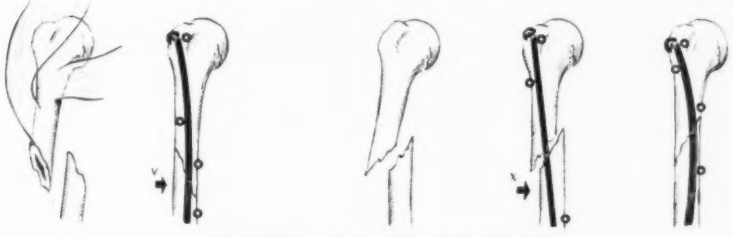
LOWER FEMUR

Double pins required. Introduce most effective pin first.

UPPER FEMUR

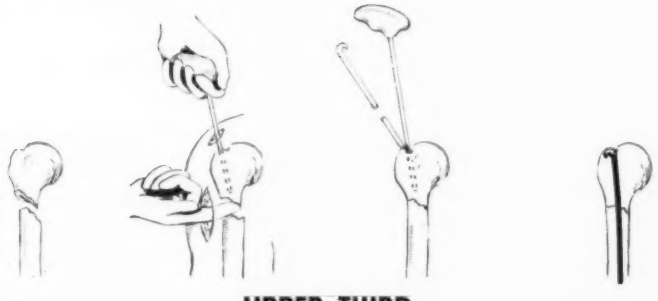
Dependent upon direction of fracture line, special type pin might be indicated.





HUMERUS, BELOW DELTOID

Requires precise handling for good healing. For fracture at right use large curved pin for compression.



UPPER THIRD

To avoid angulation, insert pin axially in short fragment.

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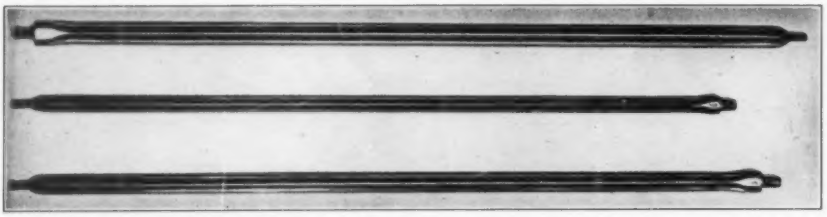

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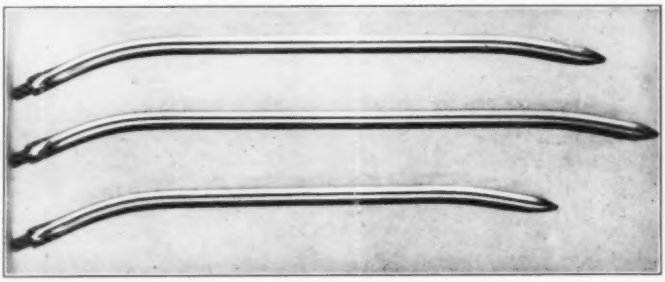

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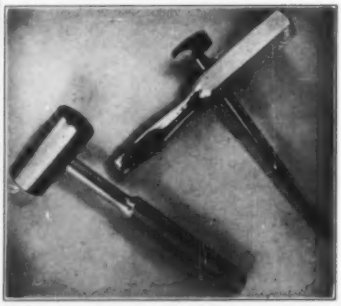

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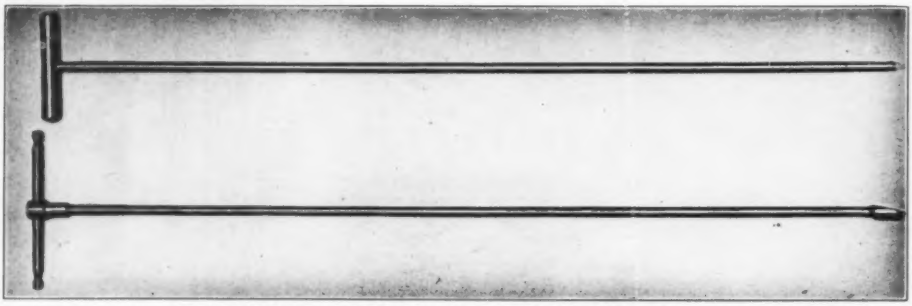

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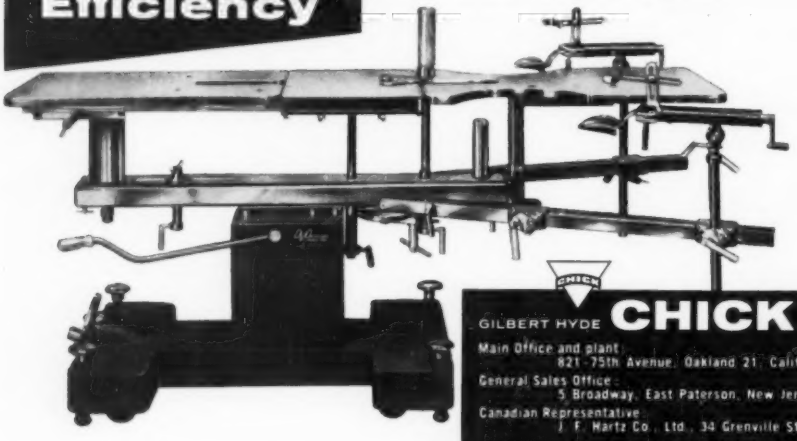

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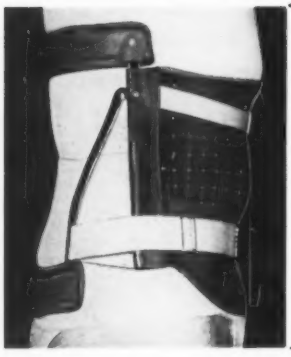

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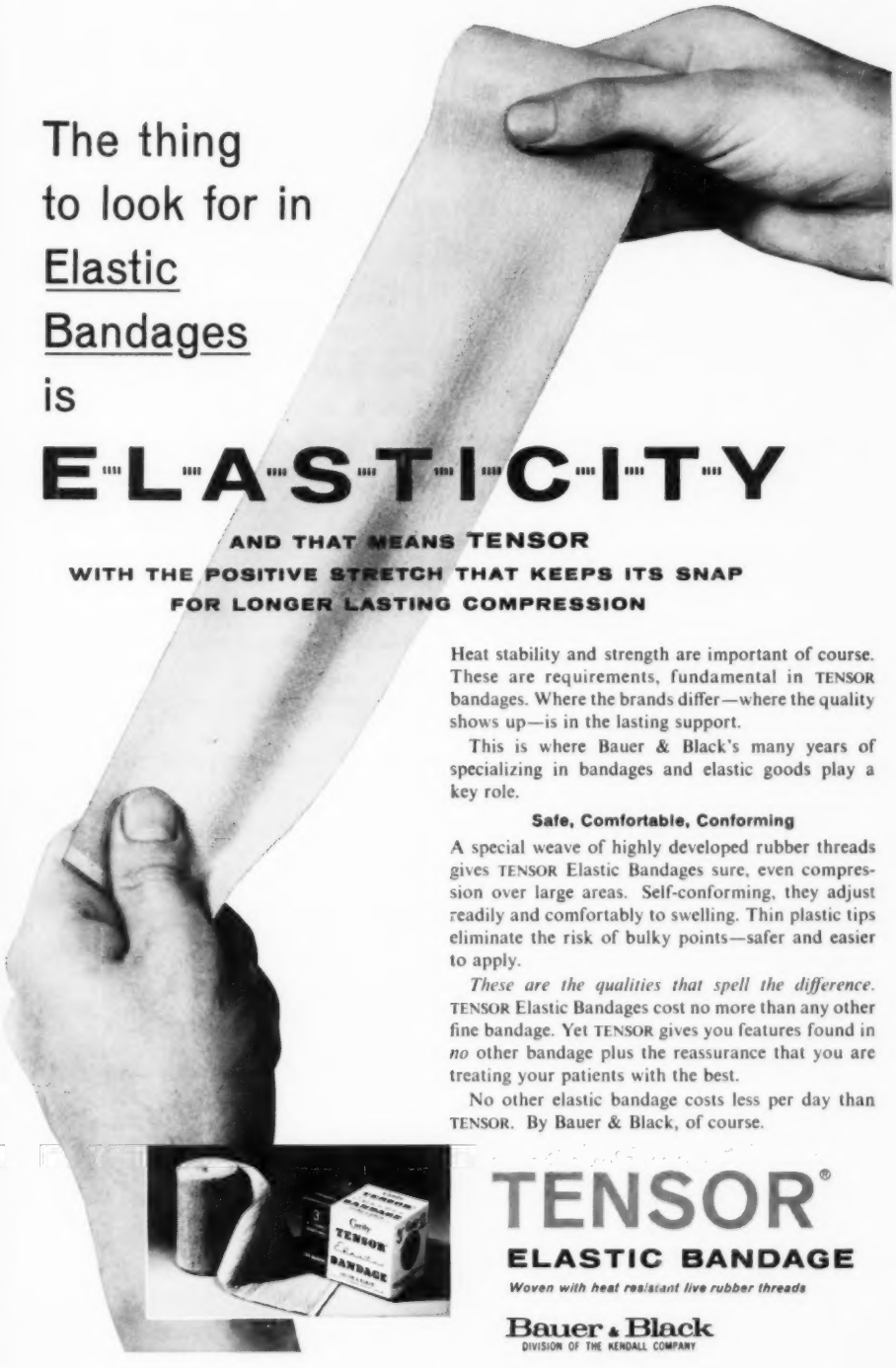

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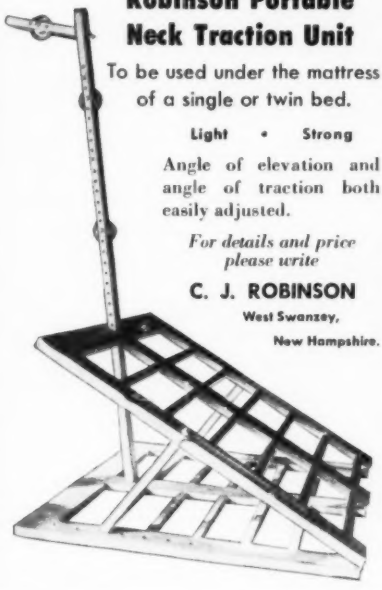

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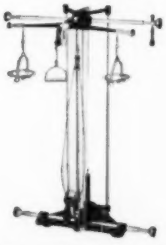

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